

**A STUDY ON**  
**SWETHA KUTTAM**

*the dissertation Submitted by*

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*under the Guidance of*

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# INTRODUCTION

”மறுப்பது உடல் நோய் மருந்தெனலாகும்  
மறுப்பது உளநோய் மருந்தெனலாகும்  
மறுப்பது இனிநோய் வாராதிருக்க  
மறுப்பது சாவை மருந்தெனலாமே”

- திருமந்திரம்

The word siddha comes from the word siddhi which means a perfection or heavenly bliss.

The masters of this ancient medical science are called as ‘Siddha’s. They are greatest spiritual scientists who are highly cultured intellectuals with spiritual insight and personal discipline.

These siddhars evaluated several approaches (Noi Nadal) and methods of preparation of medicine to provide an optional health and symphony of life to the patient .

Siddhars formulated the drugs through their spiritual intuition, not through their experiments with animals or humans

According to Siddhar’s, medicine is defined as the combination of substances or methods that cures physical and mental ailment and prevent diseases to help mankind to live immortal life.

Siddha system clearly underlines the classic concept of “Pancha Bootha System”

1. The Macro cosum (Universe)
2. The Micro cosum (Body)

”அண்டத்தில் உள்ளதே பிண்டம்”

They hold that the universe is a macro cosum made up of 5 pridominal elements or “Pancha Boothas” viz Nilam (earth), Neer (Water), Neruppu (Fire), Vali (wind) and Veli (Space) and the human being is a micro cosum made up of these 5 elements.

When there is a change in equilibrium of this 5 elements in macro cosum it influence the equilibrium of 5 elements in micro cosum. This imbalance leads to the course of a diseases.

In the present universe, the environment is highly disturbed (imbalanced). The air is polluted, water is contaminated, and food is adulterated. Also the human mind is adulterated at a will. These changes cause several diseases in humans.

One such the disease the human are suffering is Swetha Kuttam in modern medicine Swatha Kuttam is related to Leucoderma or Vitiligo.

Even before several centuries siddhar yugi as classified skin disease under a generic name called “Kuttam” as 18 types. Swetha Kuttam is one of them.

- Vitiligo affects 1% of the population
- Common in all races, predominant in dark skin
- The sex incidence is equal
- Onset, usually between 10 and 30 years of age
- The course is unpredictable
- About 30% of patients give a family history.

The author has selected to study the compound “Puvarasam Pattai Kudineer Chooranam” as internal medicine in the treatment of swetha kuttam.

The author has also selected the compound “Nuna Thailam” as external application in the treatment of swetha kuttam.

It is to be noted that the author has selected “Herbs” (Mooligai) as the first choice of medicine, when compared to metal (Thathu) minerals of animal origin (Seevan).

It is because the author has a strong belief in the saying,

”வேர்ப்பாரு தழைப்பாரு மிஞ்சினக்கால்  
பற்ப செந்தூரம் பார்”

The intention of this study of the author is to educate, help, treat and uplift the lives of patients suffering from swetha kuttam. Let the God hear the voices and cries of the suffering.

## AIM AND OBJECTIVES

### **Aim:**

About 20 to 30% problems in human being pertain to dermatology, lot of interest has been developed in this field. Hence this study was carried out with an intention to study the “Swethakuttam” in various aspects with modern comparison and to formulate an apt treatment that would be given to the patient through Siddha Medicine.

### **Objectives:**

This Scientific work on Swethakuttam was carried out to assess:

1. The incidence of this disease with age, gender, occupation, social, status diet, seasonal variation.
2. To collect the review of the disease dealing with definition, etiology, classification, signs and symptoms, prognosis, treatment and diet for Swethakuttam.
3. To correlate the signs and symptoms of Swethakuttam with that of modern science.
4. To expose siddha diagnostic principles.
5. To find out the quality safety and efficacy of the trail drug by doing
  - Chemical Analysis
  - Acute and sub acute toxicity Studies
  - Pharmacological Studies
6. To find out the side effect or adverse reactions if any,
7. To Evaluate the Clinical efficacy of trail drugs.



# **REVIEW OF LITERATURE**

## **SIDDHA ASPECTS**

### **Swetha Kuttam**

#### **(Swetha – White)**

Swetha Kuttam is a Chronic Skin disease Characterized by various sizes of hepato pigmentary patches in the skin.

According to Yugi Munivar classification Swetha Kuttam is one among the 18 types of Kuttam.

தோல் பிணிகள் 18ல் யுகிமுனி பகுப்பின்படி சுவேதகுட்டம் ஆனது

“தடிப்பான தவளநிறம் போல் வெளுத்துச்

சங்வாங்கமும் வெளுத்தாற்றான் நிரும்பும்

மடிப்பாக மயிர்வெளுத்தா லசாத்யமாகும்

வரிவுதடுதள் ளங்கைக்குதங்குய்யந்

நெடிப்பாக நெருப்புபட்டது போற்புண்ணாய்

நிறமிருந்தா லசாத்தியமென்றே யுரைக்காலகும்

வெடிப்பாக மேனியெல்லாம் வெளுத்து வீங்கில்

வெண்சுவேத குட்டமென்றே விளம்பலாமே”

-யுகி சிந்தாமணி – 800(பக்கம் 198)

- தவளநிறம் போல் வெளுப்பு
- உதடு உள்ளங்கை நெருப்புபட்டது போல் புண்
- மேனி எல்லாம் வெளுத்து வீங்கல்
- மயிர் வெளுத்தல்

Siddha literatures states that if the lesions are present in lips, palms soles anal and genital region are incurable.

Pigmentary disease are common and particularly distressing to those with darker skin.

**நோய் வரும் வழி (Aetiology)**

**Aetiological Analysis of Siddha literature**

**Thirumoolar Karukkidai Vaithiya Nool**

வியாதியுள் மூவாறு விளங்கிய குட்டங்கேள்

சுயாதிக் கிரந்தி கழன் மேகத்தாலும்

புயாதி மண்ணுளிப் பலவண்டி னாவெலட்டு

நீயாதிப் பழுநாலாய் நின்றதிக் குட்டமே”

- திருமூலர் -

- Six types of kuttam, are caused by kiranthi noi Meha Noi
- Eight types are caused by Inserts in the soil (மண்ணுளிப்புழு, வண்டு)
- Four types are caused by worms

### According to yugi vaidhya Chindhamani 800

“விளம்பவே மிகந்த உஷ்ணந்தன் னாலும்  
மிகுந்த சீதளத்தாலு மழற்சி யாலும்  
வளம்பவே மந்தத்தால் வாந்தி யாலும்  
மகத்தான பெண்ணோடு மருவலாலும்  
கிளம்பவே கிலேசங்கள் மிகுதலாலும்  
கெடியான வறக்கங்கள் டைவதலாலும்  
தளம்பவே மயிருகற்கள் தவிடு மண்கள்  
சாதத்திற் பருகலால் மிகுக்குங் குஷ்டம்”

பக்கம் எண் 199

The above said literature clearly indicates that the predisposing factors of the diseases are

- Excessive heat and cold, allergy
- Vomiting due to indigestion
- Hyper sexual indulgence.
- Mental disturbance
- Excessive sleep in day time
- Frequent intake of food mixed with polluted stone husk and hair

குட்டந்தான் பதினெட்டு வரவே தென்னிற்  
குருநிந்தை சிவநிந்தை மறையோர் நிந்தை  
திட்டந்தான் தேவையைத் தூஷணைக்கு ரோதம்  
செப்பலாற் றிருடலாற் பரதாரத்தை  
அட்டந்தா னாசையால டைக்க லத்தை  
அபகரித்த லகதிபர தேசி தன்னை  
வட்டந்தான் வைதாலார் கற்பழித்தல்  
வந்திடுமே பதினெட்டு குட்டந்தான்

-பக்கம் எண் 199

- Use of indelcent and disrespectful words against God and highly Religious and noble people.
- Intention to spoil others
- Raping
- Greed
- Cursing the elders and soon have also been given as predisposing causes by yugi test

These habits are supposed to be the factors which lower the immunity of the body (Eyarkkai vanmai) and make it vulnerable to the disease.

#### **According to Agasthiyar Kanma Kandam**

சேர்ந்த குட்டமோடு குறைநோய்கள் வந்த  
 சேதி கேள் மலராத வரும்பு கொய்தல்  
 தாரிந் சீவசெந்து வதைகள் செய்தல்  
 தாய்தந்தை மனது நொந்து ரோந் தானே  
 தானென்ற தெய்வரு தனையழித்தல்  
 சார்வான பெரியோர்கள் தமைப் பழித்தல்  
 கானென்ற நந்தவனம் பூஞ்செடிகள் வெட்டல்  
 கருமமடா சரீ ரத்திற் காசுபோலே  
 ஊனென்ற வுடம்பெல்லாம் பொட்டு பொட்டா  
 யுடன் வெளுத்து குறைநோயுதிரஞ் சிந்தும்  
 வானென்ற கருமங்கள் தீர்ப்பதற்கு  
 வகையொன்னு சொல்வேன் கேள்

-பக்கம் எண்-214

மலராத அரும்பு கொள்தல், சீவ செந்துகளை வதைகள் செய்தல் பெற்றோர் மனதை நொகும்படி செய்தல் பெரியோர்கள் தம்மை பழித்தல் நந்தவனம் பூஞ்செடிகள் வெட்டுதல் ஆகிய இக்காரணங்களால் குட்டநோய் வரும் என்று அகத்தியர் கன்ம் காண்டத்தில் கூறப்பட்டுள்ளது.

சித்த மருத்துவ சிறப்பு – நூலின்படி:-

1. தோலில் துணி முதலியவற்றால் விடாமல் ஏற்படும் உராய்வு
2. தூம்பற்ற சுரபிநீர்களின் விகற்பம்
3. நெகழ் பொருள்கள் தோலில் அடிக்கடி உராய்தல்
4. சத்தற்ற உணவு
5. இந்நோய் ஒருவகை காமியத்தால் பிறக்கும்
6. வில இம்மிகள் குறைந்து போதல் (அ) இல்லாமல் இருத்தல்
7. மேகநோய், கொறுக்கு நோய்க்கு துணையாகவும்
8. தொழுநோயிலும் வெண்படை காணும்
9. மேகநோயிலும், தொழுநோயிலும் வரும் வெண்படை தொற்றும் மற்றவை தொற்றாதவை

As analysed in Siddha system predisposing causes for this disease varies from hereditary, factor, stress and strain, mal nutrition and venereal exposure, no other specific causes have been mentioned for Swethakuttam.

- அழுகிய மீன், நண்டு, நத்தை, சிப்பி, இவைகளை மிகுதியும் உண்டபின் யோகநிலையில் இருத்தல்
- வேகா உணவுகளை கொள்ளல், வயிறு நிறைய உண்ட பின் யோகநிலையில் இருத்தல்.
- தன்வினை, பழவினை, தாய்
- தந்தை வழியாகவும் பிறக்கும்

## Classification:

According to Yugi Vaidhya Chinthamani

முத்தாகும் குட்டந்தான் பதினெட்டுக்கும்  
முனியான யுகிநான் சொல்லக் கேளாய்  
புத்தாகும் புண்டரீக குஷ்டத்தோடு  
பொருகின்ற விற்போடகக் குட்டமாகும்  
பத்தாகும் பாமா குஷ்ட ஏகசர்ம குஷ்டம்  
பரிவான கர்னகுஷ்டம் சர்மகுஷ்டங்  
கித்தாகுங் கிருஷ்ண குட்டம் அவதும்பர குட்டம்  
கேடியான மண்டல குஷ்டமாகு மென்னே  
குட்டமா பரிசு குஷ்ட மோடு  
மருவலாங் கிப குஷ்டந் சர்மதல குஷ்டந்  
திட்டமாந் தத்துரு குஷ்ட மோடு  
தக்கான சித்துமா குஷ்டஞ் சதாரு குஷ்டந்  
துட்டமாஞ் சுவேத குஷ்டதன் னோடொக்கச்  
சுயம்பான பதினெட்டுக் குட்டமாச்சே

-பக்கம் எண்: 191

1. Pundareeka kuttam
2. Virpodaka kuttam
3. Baama kuttam
4. Gaja saruma kuttam
5. Karna kuttam
6. Sarma kuttam
7. Krishna kuttam
8. Avudhumbara kuttam
9. Mandala kuttam
10. Abarisa kuttam
11. Visarchika kuttam
12. Vibaathika kuttam
13. Kideeba kuttam
14. Sarmathala kuttam
15. Thethru kuttam
16. Sithuma kuttam
17. Sathaaru kuttam

18. Swetha kuttam  
Swetha Kuttam is also called as Venpadai.

**1. According to Siddhar Aruvai Maruthuvam**

**Venpadai has been classified into 3 types on the basis of Mukkutram they are,**

1. Vaatha Venpadai
2. Pitha Venpadai
3. Kaba Venpadai

**Clinical Features:**

1. The Skin appears glittering and tough
2. There is an excessive perspiration or no perspiration
3. Discolouration
4. Heat and itching of the skin
5. Numbness in some parts of the body

**2. According to Sirappu Maruthuvam**

1. Vaatha Venpadai
2. Pitha Venpadai
3. Kaba Venpadai
4. Mega Venpadai

**(i) Vaatha Venpadai**

It is characterized by the depigmented patches, which are dry, rough and reddish with somewhat pale-black colour.

**(ii) Pitha Venpadai**

It is Characterized by the depigmented patches red in colour like lotus flower, spreading with burning sensation and loss of hairs on that area.

### **(iii) Kaba Venpadai**

It is characterized by the depigmented patches white in colour like leucus flower, spreads with rashes and itching.

### **(iv) Mega vepadai**

It is due to the venereal diseases and it occurs after 4 or 6 months after the onset of disease, syphilis within four or six months of the attack. This venpadai develops initially along the nape and the adjoining spaces. Also gradually it affects the shoulder joints, back of trunk. Clinical features of this type are clearly defined by the author of Siddha Maruthuvam Sirappu” as follows:

Depigmented patches are small in number, pale in colour, with turmeric colour or dark colour margin marked with hyperpigmented signs. These lesions are circumscribed with 2mm to 3mm diameter or above. This correct picture of hypopigmented and hyper-pigmented skin seems to be more or less a multi eyed filter (sieve-like).

Females are more prone to this Mega Venpadai. Therefore anti syphilitic therapy is mandatory in the early period of the treatment.

### **According to Athma Rakshamirtha Vaidhtya Sarasankiraham**

Swethakuttuam is classified into 4 types

1. Venkuttam
2. Senkuttam
3. Karunkuttam
4. Peru Viyathi



## Clinical Features of Kutta Rogam

### 1. According to Agasthiyar Ayurvedham 1200

“உரைத்தார் பதினேழங்குட்டமுற்ற சரீரமுடல் வெளுக்க  
லிருந்தும் பதினெட்டாங்குட்ட மிதமாய் தொண்டைத் தானகட்டும்  
பெருத்த சரீரம் வெள்ளை பற்றிப் பெருக்க துயரமுண்டாகும்  
வருத்தங்காணும் பதினெட்டாம் வகையாங் குட்டவகை சொன்னோம்”  
- பக்கம் எண்:235

### 2. According to Thanvanthiri Vaithyam

“மீக்கெளத் தோறுமெலுமோர் முகம் வெளுக்குமாகில்  
நோக்கியல் மரிக்குஞ் சொன்ன வெண்குட்டமாமே”  
-பக்கம் எண்:319

### 3. According to Vaidhya Saarasangiraham

- \* Sole, hands, lips, scalp, fingers and wrist joint – all organs are found with white coloured patches which are circumscribed along with thickened border and gradually spread which is known as “Swetha Kuttam”.
- \* Blood, muscle and adipose tissue are affected by disease.
- \* Dislocation of hairs, absence of normal skin texture comparing the adjoining normal skin area and appearance of burns is not curable.

### 4. According to Anubhava Vaidhiya Deva Ragasiyam

இந்நோயை குஷ்டமென கூறினும் இது குஷ்ட வகைகளினின்று வேறுபட்டது என்பதையும் குஷ்டத்தைப் போல் அவ்வளவு கொடுமையான வியாதி அல்லவென்றும் உணர வேண்டும். இந்நோயில் திட்டு திட்டாக வெண்மை நிறமான படைகள் உண்டாகி பிறகு தேகம் முழுவதும் பரவி உடலை விகாரப்படுத்துதல் குணங்களை உடையது”.

### **Character of Swetha Kuttam**

Skin colour will change to reddish black or reddish white or white colour with spreading nature. The imbalance of the three thathu produces certain lesions in skin known as kuttam.

Absence of perspiration and thickening of skin may produce the colour changes in skin.

தீரும், தீராதவை

### **In Thanvanthiri Vaithiyam**

சாத்தியம் - 11

“பூண்டந் நூரவினோடு சதாரிகம் புண்டரீ கந்த

தாண்டு விற்போடம் பாமாவுடன் மைதலம் வெண்குட்டம்

கூண்டிடு காகறந்தி சிறுமை யசல குட்டம்

வேண்டிய வியாதியோடும் பதினொன்றும் விரித்துக் காணே”.

-பக்கம் எண்:325

அசாத்தியம் - 7

“சொல்லுகுட்டம் ஏழுவகைபேர் சொல்லிக் கபால சர்மீகம்

வெல்லு முதும்பா மேகிடிபம் விசர்ச்சிமண்டலக் கிரமும்

மல்லல் தருமீசி யகுவை யாகும் பெயரோ ரேழாகும்

வல்லகியாதிக் குணமதனை வகுத்தப் பாரிலுறுரைப்பேனே”.

-பக்கம் எண்: 321

### Curable -11

1. Thethuru Kuttam
2. Sadhaaru Kuttam
3. Pundareega Kuttam
4. Virpodaga Kuttam
5. Sama thala Kuttam
6. Baama Kuttam
7. Kaha Nandhi Kuttam
8. Venkuttam
9. Sithuma Kuttam
10. Alasakuttam
11. Vibaathiga Kuttam

### Incurable -7

1. Kabaala Kuttam
2. Sarumamega Kuttam
3. Avudhumbara Kuttam
4. Kideeba Kuttam
5. Visarchika kuttam
6. Aguvai Kuttam
7. Mandala Kuttam

### In Yugi Vaidhya Chinthamani -800

குட்டந்தான் பதினெட்டில் சாத்தியந்தான்  
கூறக்கேள் விற்பொடக பாமா குட்டம்  
திட்டந்தான் கெசசர்ம குட்டமொடு  
கிருட்டின குட்டமவதும்பர குட்டந்தானும்  
திட்டமாந் தேதிதிருக் குட்டமொடு  
செய்சித்துமா குட்டங் கிடிப குட்டம்  
தட்டந்தான் மிகுந்த சதாரு குட்டம்  
சமகிருட்டண குட்டம் சாத்தியமா மென்னே”.

-பக்கம் எண்:200

### **Curable-10**

1. Virpodaka kuttam
2. Baama Kuttam
3. Gaja Saruma Kuttam
4. Krishna Kuttam
5. Avuthumbara Kuttam
6. Thethuru Kuttam
7. Sithuma Kuttam
8. Kideepa Kuttam
9. Sathaaru Kuttam
10. Sarmathla Kuttam

### **Incurable-8**

1. Pundareeka Kuttam
2. Karna Kuttam
3. Sikura Kuttam
4. mandala Kuttam
5. Abarisa Kuttam
6. Visarchika Kuttam
7. Swetha Kuttam
8. Vivadhika Kuttam

According to Siddha literature if the lesions are present in palms, soles, anal region, genitalia area incurable,. The patches look like burns is also incurable.

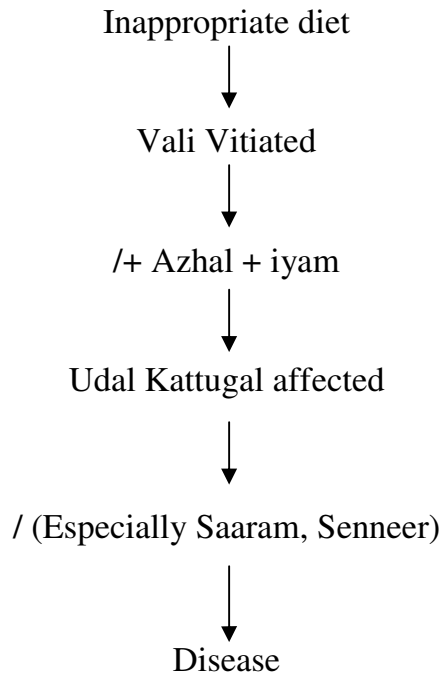
### **Mukkuutra Verupadugal (Pathogenesis):**

“வாதமலாது மேனி கெடாது”

- தேரையர்

- நோய் நாடல் நோய் முதல் நாடல் திரட்டு

- பக்கம் எண்:688



### **THINAIGAL – LANDS (GEOGRAPHICAL AREA)**

Geography affects a person in a same manner as the seasons. Nilam is classified into 5 types, depending upon the surrounding, Vegetation, landscape and ecological state.

Study of the five lands is very necessary, because certain diseases are vulnerable in certain lands.

<b>S.No.</b>	<b>Thinaigal / lands</b>	<b>Geographical area</b>	<b>Common diseases</b>
1.	Kurinji – Hilly trea	Mountains & its Surroundings	Iya noi, Liver Diseases
2.	Mullai – Sylvian Tract	Forest its Surroundings	Azal, Vali, Liver Diseases
3.	Marutham – Fertile area	Fields & its Surroundings	Ideal place for healthy Living
4.	Neithal – Coastal Area	Sea & its Surroundings	Vali diseases, Liver Diseases
5.	Paalai – Arid area	Desert & its Surroundings	Vali Azhal Iyam Diseases

Each region has got its own characteristics features which influence the inhabitant's mental, physical, economic, occupational and cultural activities. In each region, on the basis of its peculiar physical and climatic features, some ailments are endemic. The preventive and curative measures for these ailments are stated in the medical literature.

### **PARUVAKALAM /( SESONAL EFFECTS):**

Because of the climate changes, which are the results of solar, lunar and planetary effects, natural changes take place in the earth and its atmosphere as well as human body. A certain change in the aggravation, displacement of aggravation or normal sign of Mukkutram during different seasons in normal and the biological processes in the body become adapted to these changes.

SIDDHARS described six seasons in a year and each constituting two months some of the diseases are more prevalent during the particular Paruvakalam (season) and study of it will be of much use of diagnosis.

**Here is a table of the Principle seasonal effects:**

<b>S.No.</b>	<b>Kaalam</b>	<b>Synonym</b>	<b>Kutram</b>	<b>Suvai</b>
1.	Kaar Kaalam (Aug 16 –Oct 15)	Early Rainy	Vali Azhal ↑	Inippu, Pulippu, Uppu
2.	Koothir Kaalam (Oct. 16 – Dec.15)	Late Rainy (Autumn)	Vali (-) Azhal ↑	Inippu, Kaippu, Thuvarppu
3.	Munpani Kaalam (Dec.16-Feb.15)	Early dew (Winter I Part)	Azhal (-)	Inippu, Pulippu, Uppu
4.	Pinpani Kaalam (Feb.16 – April 16)	Late dew (Winter II Part)	Iyam	Inippu, Pulippu, Thuvarppu
5.	Elavenil Kaalam (April 16 –June 15)	Early Summer	Iyam ↑↑	Kaippu, Karppu, Thuvarppu
6.	Muthuvenil Kaalam (June 16- Aug. 15)	Later Summer	Vali ↑ Iyam (-)	Inippu

- ↑ Accumulation (Thannilai Valarchi)
- ↑↑ Aggravation (Vetrunilai Valarchi)
- (-) Alleviation (Thannilai Adaithal)

In every season there will be changes in the land, water, plants, animals and human beings, which will modify the physiology and making (rendering) them susceptible to certain specific disease which are common in these seasons. The siddhars have been anticipating these changes and advised certain measures in the form of diet, purgative exercises, etc, to avoid the onset of such ailment.

### **UYIR THATHUKKAL / MUKKURTRAM:**

These are all the main three pillars which functions the body with an equilibrium state. Any disturbance in that state leads to diseased condition in our body.

#### **The three pillars are**

1. Vali
2. Azhal
3. Iyam

#### **(i) VALI or VAYU:**

Vali is not mere wind, but also that which causes motion, energy and sensation of every cell in the body. Vayu relates to nerve force. It is responsible for all movements in the mind and the body. In human body it controls the Gnanedriyam (sensory actions) & Kanmendriyam (motor activities).

#### **Vali generally lives in:**

Abaanan, Edakalai, kamakodi, Undhiyin Keezh Moolam, Hip region, Bones, Muscles, Nerves, Joints, Skin, Hair follicles, Stools.

## **Varieties of Vali:**

According to their location and functions they are classified into 10 types.

1. Uyirkkal (Pranan)
2. Kezhnökkum kaal (Abaanan)
3. Parravukaal (Viyanan)
4. Melnökkum Kaal (Udhanan)
5. Nadukkaal (Samanan)
6. Naagan
7. Koorman
8. Kirugaran
9. Devadhathan
10. Thananjeyan

### **1. Uyirkkaal (Pranan) (Heart Centre)**

It regulates the respiratory, cardiac and digestive system. By joining with pingalai, it forms azhal naadi. It is responsible for bio confusion in the body.

In Swetha Kuttam, Uyirkkaal may be affected.

### **2. Kezhnökkum Kaal (Abaanan) Mooladharam Centre)**

It regulates the defaecation, micturation, menstruation, parturition and ejaculation. It corresponds to the pelvic plexus and the lower part of the gut.

### **3. Paravukaal (Viyanan) (fore head centre)**

It spreads all over the body and all nerve endings. It regulates constriction and relaxation of the voluntary and involuntary muscles. The neurological problems were due to this Vayu. It spreads the nutrients to all over the body from the digested food.

### **4. Mel Nokkumkaal (Udhanan)**

It is responsible for speech, vomit, hiccough and sneeze.



**5. Nadukkaal (Samanan):**

It is responsible for digestion and it spreads the nutrients all over the body. Joining with suzhumunai forms the Kaba naadi. It neutralizes the other four Vayus.

**6. Naagan:**

It is responsible for the intelligence and derangement of this Vayu causes Impaired memory. It helps to opening and closure of eyelids.

**7. Koorman:**

This is responsible for the vision, Yawning and Lacrimal secretions.

**8. Kirugaran:**

It is responsible for salivation, nasal secretion, hunger, sneeze, cough and concentration.

**9. Devadhathan:**

It is responsible for laziness and anger.

**10. Thananjeyan:**

It produces swelling all over the body and leaves from cranium only after the 3<sup>rd</sup> day after death. It is responsible for the decay of the body after death.

**II. AZHAL:**

This is nothing but the characteristics of fire such as burning, boiling and heating etc. It corresponds to the functions of i.e. thermogenesis production of heat necessary to maintain integrity of the human circulatory system. Azhal is classified into 5 types. It mainly governs enzymes & hormones.

**Seats of azhal:**

Between heart & the navel, Sweat, lymph, blood, stomach, urinary bladder, saliva eye and skin.

S.No.	Name	Location	Function
1.	Akkuanal (Analagam)	Stomach Small intestine	Dissolvent & Digestive
2.	Vanna eri (Ranjagam)	Liver, Spleen, Stomach	Colouring, Pleasin, Gratifying
3.	Attralangi (Sathagam)	Heart	Effective efficient
4.	Nokku Azhal (Alosagam)	Eyes	Seeing, consideration
5.	Ollolithee (Prasagam)	Skin	Complexion of the skin

**In Swetha Kuttam, Vannaeri (Ranjagam), Ollolithee (Prasagam) were affected.**

**III. IYAM**

It imparts moisture. Iyam is located in samanana semen, head, tongue, flat, bone marrow, blood, nose, chest, nerves, brain, large intestine, eyes, stomach & pancreas.

S.No.	Name	Location	Function
1.	Alli Iyam (Avalambagam)	Lung	Supports all the other
2.	Neerpi Iyam (Kilethagam)	Stomach	Moistens and nourishes the food
3.	Survaikanna Iyam (Pothagam)	Tongue	Take care of perception
4.	Niraivu Iyam (Tharpagam)	Head	Refrigerant effect to eyes
5.	Ondri Iyam (Santhigam)	Joints	Stability, Lubrication & movements of joints

Since Swetha kuttam patients are not having defined description of pathology, the pattern of disturbance in Vali, Azhal, iyam keeps on varying. The Manifestation of these uyir thathus keep on changing according to the predominant symptom. But alteration in azhal thathu is seen mostly.

## **UDAL KATTUGAL : (SEVEN PHYSICAL CONSTITUENTS)**

### **1. Saaram – Chyle (Plasma)**

It is responsible for the growth & development. It keeps the individual in good spirit and nourishes the blood.

**In Swetha Kuttam, Saaram may be affected.**

### **2. Senner – Blood**

Blood imparts colour to the body and nourishes the muscle for the ability.

**In Swetha Kuttam, Senneer may be affected.**

### **3. Oon – Muscle:**

Gives shape to the body.

### **4. Kozhuppu – Fat:**

It helps in lubricating the different organs and maintains oily matter of the body.

### **5. Enbu – Bone:**

It supports the system and responsible for the posture movement of the body.

## **6. Mulai - Marrow**

It fills the bone cavity, nourishes semen and imparts strength, endurance and shiny appearance.

## **7. Sukkilam or Suronitham (Sperm & Ovum):**

It is responsible for the reproduction.

### **RELATIONS BETWEEN SEVEN UDAL THATHUS & MUKKUTRAM**

<b>S.No.</b>	<b>Udal Kattugal</b>	<b>Governing Kutrams</b>	<b>Bootham</b>
1.	Saaram (Plasma)	Iyam (Kabam)	Neer
2.	Senneer (Blood)	Azhal (Pitham)	Thee + Neer
3.	Oon (Muscle)	Iyam (Kabam)	Mann + Neer
4.	Kozhuppu (Fat)	Iyam (Kabam)	Mann + Neer
5.	Enbu (Bone)	Vali (Vatham)	Mann + Vali
6.	Mulai (Marrow)	Iyam (Kabam)	Neer + Vali
7.	Sukkilam/Suronitham (Reproductive tissues)	Iyam (Kabam)	Neer

### **PINIYARI MURAIMAI – (DIAGNOSIS)**

The methodology of diagnosing in siddha science is very unique and solely based on the clonical acumen of the physician. It is based on the three main principles.

1. Poriyal Aridhal
2. Pulanal Aridhal
3. Vinaathal

“Pori” is the five organs of perception namely Nose, Tongue, Eyes, Ears and skin.

“Pulan” is the five objects of senses smell, taste, vision, auditory and sensation respecti vely corresponding to “Pori”. Poriyalarithal and Pulanal Therthal go hand

in hand with the concept to examining the patients “Pori” and “Pulan” with that of the “patient’s Pori and Physicians Pulan”.

### **Vinaadhal (Interrogation)**

The physician should interrogate about the patients name, age, occupation, diet, habits, and nativity, socio economic status, if prone to any allergens, complaints, History of previous and present illness, family and personal history, duration of illness.

### **Enn Vagai Thervugal (Eight diagnostic methods in Siddha):**

It is the unique and special method in Siddha.

“நாடிப் பரிசம் நாநிறம் மொழிவிழி

மலம் முத்திரமிவை மருத்துவராயுதம்”

“மெய்க்குறிநிறம் தொனி விழிநா இருமலம் கைக்குறி”.

- தேரையர்

- நோய் நாடல் நோய் முதல் நாடல்

“தொகுக்கலுற்ற அட்டவிதப் பரிட்சை தன்னை

துலக்கமுறும் பண்டிதரே தெளிவாகப்

புகர்கின்ற வார்த்தையைப் பார் நாவைப்பாரு

வகுக்கரிய தேகமென தொட்டுப்பாரு

வனமான சரீரத்தின் நிறத்தைப்பாரு

சகிக்கிரிய மலத்தைப் பாரு சலத்தைப்பாரு

சார்ந்தவிழி தனைப் பார்த்து தெளிவாய்க்கானே”

-கண்ணுசாமி பரம்பரை வைத்தியம் பக்கம்-1

Envagai thervugal is the speciality of Siddha diagnosis. These are the instruments for the physician to diagnose disease.

### 1. Naadi:

The three Uyir thaadukkal felt through the pulse is called Naadi.

Naadi		Vayu	Uyir thathu Ratio
Edakalai	+	Abaanan	Vatham – 1
Pinkalai	+	Piranan	Pitham – ½
Suzhumunai	+	Samanan	Kabam – ¼

“கரிமுகனடியை வாழ்த்திக்

கைதனில் நாடி பார்க்கில்

பெருவிரலங் குலத்தில்

பிடித்தடி நடுவே தொட்டால்

ஒரு விரலோடில் வாதம்

உயர் நடுவிரலில் பித்தம்

சிருவிரல் மூன்றிலோடில்

சிலேத்தும் நாடி தானே”

-அகத்தியர் நாடி

-நோய் நாடல் நோய் முதல் நாடல்

### 2. Parisam:

Observations by touch, temperature, sensory impairments, masses, nodes, swelling, and texture of the skin pain, hardness, oedematous, and dullness shall be noted.

### **3. Naa:**

Signs and symptoms in the tongue are considered here. Size, appearance, thickness, colour (pigmented, magenta) fissured (longitudinal, transverse) coated, geographical patches, oral hairy leucoplakia, candida, aphthous ulcers, sense of taste, saliva secretion.

### **4. Niram:**

The colour of skin is mainly considered here but also the change in other organs.

**In Swetha Kuttam, Niram was affected.**

### **5. Mozhi:**

The change in the normal sound of voice mainly urattha oli (Valithel), thazhltha oli (Melithal), physiological and mental status can also be noted during conversation.

### **6. Vizhi**

Colour, warm, burning sensation, irritation, visual perception.

### **7. Malam:**

Nature, quantity, colour, odour, froth, consistency are noted.

### **8. Moothiram:**

The urine examination classified into two types

#### **a) Neerkkuri**

“வந்தநீர் கரி எடை மணம் நுரை எஞ்சலென்”

-தேரன்

-நோய் நாடல் நோய் முதல்நாடல்

Urine is to be observed for the following characters

Niram (colour)

Edai (specific gravity)

Manam (smell)

Nurai (froth)

Enjal (deposit)

**b) Neikkuri**

It is an important test to assess the predominantly affected humour.

அருந்துமா நிரதமும் அவிரோதமதமாய்  
அஃகல் அலர்தல் அகாலவூண் தவிர்த்தழி  
குற்றவருந்தி உறங்கி வைகறை  
ஆடிக்கலசத் தாவியே காதுபெய்  
தொரு மகூர்த்தக் கலைக் குட்படு நீரின்  
நிறக்குறி நெற்குறி நிருமித்தல் கடனே

-தேரன்

-சித்த மருத்துவாங்க சுருக்கம் (பக்கம் எண்: 509)

On the day before the urine test one should take food, consisting of all the six tastes at the regular time based on one's digestive fire; after a sound overnight sleep, urine should be collected in glass ward and the test should be done before 90 minutes from dawn.

A drop of oil is dropped at the centre of urine bowl without any shake. It should be ensured that the Sunlight falls on it, but is not disturbed by the wind. A keen observation of the oil drop suggests the condition of the patient.



If the oil drop takes the shape of a snake, it indicates Vatha disease. If it spreads like a ring it indicates Pitha and if it stands like a pearl it indicates Kapha disease.

If there is a combined shape like a ring in a snake, or snake in the ring, snake and a pearl in the ring, it indicates combined derangement of humors.

Swetha kuttam starts with disturbed Azhal and eventually involves all the three uyir thathus-thus resulting in various patterns of oil spread in the urine surface.

### **NOI NEEKAM (TREATMENT)**

In siddha system the main aim of treatment is not only for the removal of physical illness but also the mental illness. Treatment is considered with prevention and improvement of the general body condition (rejuvenation) also.

This is said as follows:

Kappu/Thuvarppu	-	Prevention
Neekkam	-	Treatment – curative
Niraivu	-	Restoration – promotive

While treating the disease the following principles must be noted.

“நோய் நாடி நோய் முதல்நாடி அது தணிக்கும்

வாய்நாடி வாய்ப்பச் செயல்

“உற்றானளவும் பிணியளவுங் காலமுங்

கற்றான் கருதிச் செயல்”

-திருக்குறள்

So it is essential to diagnosis properly to know about the etiology, the nature of the patient, the severity of illness, the seasons and the time of the occurrence of the disease.

## LINE OF TREATMENT:

1. To bring the three Kutrams in equilibrium
2. Medicines (Int & Ext)
3. Diet and advises

### 1. Bring the three Kutrams in Equilibrium:

Since Siddha system of medicine is based of Mukkutra theory, the purgation (kalichal Maruthuvam) was given by for the vitiation of three humours.

Agasthiyar Kuzhambu 100mg was administrated at early morning as a purgative in the prior day of treatment.

### 2. மருந்துகள்

உள்மருந்து	:	பூவரசு பட்டை குடிநீர் சூரணம்
அளவு	:	25கி இருவேளை 60மிலி
புறமருந்து	:	நுணா தைலம்

### 3. Diet and Advices

#### Recommendations

- Use Copper rich water storing it in a copper vessel for at least 8 hrs.
- Increase green vegetables, carrot, bottle gourd, beans & pulses intake.
- Consume soaked red grains 30-50gms / day as an immunomodulator.
- Deworming is necessary in case worm's infestation.

**Restrictions**

- (1) Citrus foods viz. Lemon, Orange, Grapes, Tomato, Pickles, Mango, Fine flour (Maida).
- (2) Butter milk(lassi), Apple, Chilli< Urad dal and food containing sour and Vitamin C contents.
- (3) Spicy, Salty Foods.
- (4) To avoid Curd, Oil, Alcohol, Sugar.
- (5) Mustard
- (6) Brinjal
- (7) Non-Vegetarian diet & fast foods.
- (8) Beverages like cold drinks.
- (9) Chemical based detergents/soaps.

## **MORDERN ASPECTS**

### **SKIN**

The Skin is one of the largest organ in the body. It has many functions, the most important of which is as a barrier to protect the body from noxious external factors and keep the internal systems interact.

Hippocrates, Father of Medicine described many skin diseases and divided them into two groups according to their exogenous or endogenous causes. He attributed the origin of disease to abnormal mixing of black and yellow bile, blood and phlegm. The theory of abnormally mixed humors played a major role in dermatology for a long time.

Dermatology is a branch of medicine dealing with the skin. Its roots reach back to antiquity. The obviously manifested skin diseases have drawn the attention of men since time immemorial.

#### **Skin Anatomy:**

The human skin is the outer covering of the body and is continuous with the mucous membranes in the region of the mouth, nose, urogenital organs and the anus. In an adult the skin surface measures 1.5- 2m<sup>2</sup> while the thickness of the skin varies from fractions of a millimeter to 4mm. The thickness of the epidermis varies from 0.006 – 0.9mm to 0.5-0.6mm. The thickness of the subcutaneous fat varies considerably. Some areas are devoid of fat while in others (on the abdomen and gluteal regions), it is several centimeters thick. The mass of skin of an adult accounts for approximately 5% while together with subcutaneous fat for about 10 to 17.7% of the total body mass.

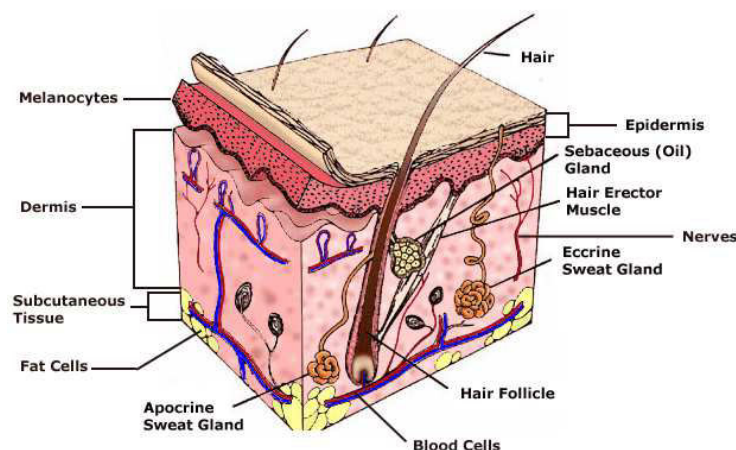
### **Skin Histology:**

The skin develops from two germinative zones. The ectoderm which is represented by the epidermis (the most superficial skin layer) and the mesoderm (the middle embryonal layer) represented by two layers namely the true skin, or dermis (the middle layer) and the subcutaneous fat or hypoderm the deepest skin layer.

The boundary between the epidermis and dermis forms a wavy line because of the presence of skin papilla (special out growth on the surface of the true skin) the spaces between which are filled with epithelial processes).

### **Layers of the skin:**

Skin is composed of three layers. The epidermis, the dermis and the subcutaneous.



### **Epidermis:**

The epidermis is defined as a stratified squamous epithelium which is about 0.1mm thick, although the thickness is greater (0.8-0.4mm) on the palm and sole. Its prime function is to act as a protective barrier. The main cell of the epidermis is the keratinocyte which produces the protein keratin.

The four layers the epidermis represent the stages of maturation of keratin by keratinocytes.

1. Basal Cell layer (Stratum basale)
2. Prickle cell layer (Stratum spinosum)
3. Granular cell layer (Stratum granulosum)
4. Horny layer (Stratum corneum)

### **Dermis:**

The dermis is defined as a tough supportive connective tissue matrix, containing, specialized structures, found immediately below and intimately connected with the epidermis. Two layers are distinguished in it, the papillary or sub epithelial layers and the reticular layer. The papillary layer is that part of the dermis which is found between the epidermis and the superficial network of blood vessels. The reticular layer merges with the subcutaneous fat and is not demarcated from it sharply. The dermis is supportive connective tissue, mainly collagen, elastin and glycosaminoglycans.

### **Subcutaneous Layer:**

The subcutis consists of loose connective tissue and fat (upto 3 cm thick on the abdomen).

### **Blood and Lymphatic Vessels:**

The skin also has a rich and adaptive blood supply. Arteries in the subcutis branch upwards, forming a superficial plexus at the papillary/reticular dermal boundary. Branches extend to the dermal papillae, each of which has a single loop of capillary vessels, one arterial and one venous. Veins drain from the venous side of the loop to form the mid-dermal and subcutaneous venous networks. In the reticular and papillary dermis there are arteriovenous anastomoses which are well innervated and concerned with thermoregulation.

The lymphatic drainage of the skin is important, and abundant meshes of lymphatics originate in the papillae and assemble into larger vessels which ultimately drain into the regional lymph nodes.

### **Pigmentation of the Skin:**

The colour of the skin may be brown or even black according to the amount of pigment present and it varies due to external and internal factors.

Even in white races most parts of the skin contain brown pigment granules in the deepest layers of the germinative zone of the epidermis. In dark races they are more abundant and extend throughout the whole zone.

The degree of racial pigmentation does not depend on the number of melanocytes present but on their metabolic activity and the size and shape of their melanin producing organelles the melanosomes.

Brownness of the skin depends upon transfer of melanosomes from melanocytes into keratinocytes. Melanosomes are cytoplasmic particle formed in melanocytes and then distributed among the basal cells of the epidermis. Each melanocyte in the epidermis secretes melanosomes are the site of melanin synthesis by the action of tyrosinase upon tyrosine.

### **Melanin:**

Melanin – Derived from the Greek word Melas, meaning black.

Melanin is complex black –brown polymer synthesized from the amino acid dihydroxyphenyl alanine (L-DOPA).

The forms of melanin exist.

- Ordinary melanin known as eumelanin and a melanin synthesized from cysteinyl DOPA with a more reddish hue, known as pheomelanin.

The initial part of melanin synthesis is catalysed by a copper –containing enzyme complex known as tyrosinase which also catalyses the transformation of L.DOPA to tyrosine.

### **Melanin Formation:**

Melanin, wherever it is found, is formed in the local cells by the enzyme tyrosinase (or) melanase. The mother substance, upon which the enzyme acts, is a tyrosine derivative (DOPA) believed to be formed in the adrenals. Melanin synthesis from the oxidation of phenylalanine or tyrosine is as follows:

1. Tyrosine → DOPA → DOPA → Quinone
2. DOPA –Quinon → 2-Carboxy 2.3 – dihydro – 5.6 – dihydroxyindole.  
→ 2-Carboxy – 2.3 – dihydro – indole – 5.6 – Quinone → 5.6 Dihydroxyindole.
3. 5.6 Dihydroxyindole → Indole -5.6 Quinone → Melanin

Melanin formation in both human and amphibian skin is augmented by the Hormone known as intermedian or melanocyte – stimulating hormone (MSH) secreted by the pars intermedian of the pituitary gland. Adrenocorticotrophic hormone (ACTH) secreted by anterior Pituitary has melanocytestimulating activity similar to MSH although to a much lower degree. In Addison's disease ACTH is secreted in a large amount and there is brownish black pigmentation of the exposed parts of the skin Eg. Hands, feet and mucous membrane.

Melatonin extract from bovine pineal gland, causes concentration of melanin near the nuclei of melanocytes in frog and as a result of this the skin becomes pale. Its role in the human is not known. MSH causes the serum copper to rise and this is accompanied by an increase in the melanin formation. Diminished formation of melanin is seen in albinism and leucoderms. In melanotic sarcoma, melanin may be found in the urine.



**Distribution:**

It is widely distributed in the body but peculiarly enough it is limited only to those structures which have got an ectodermal origin, for Eg: skin, hair, choroid coat of retina and substantia nigra of the brain.

**Functions:**

The function of melanin in the choroids is namely to covert the eye ball into a perfect dark chamber. Since nervous tissue is derived from ectoderm, the melanin in the substantia nigra may represent the vestigial remnants of the melanin in the substantia nigra may represent the vestigial remnants of the melanin forming properties. Melanin is the great protector of the skin against the actinic rays of the sun.

Melanin in keratinocytes is black and absorbs all visible light, UVR and infrared radiation. It is also a powerful electron acceptor and may have other protective functions which as yet have been poorly characterized.

**Abnormal Pigmentation**

Normal pigmentation of the skin is influenced of the amount and depth of melanin, by the degree of vascularity, by the presence of carotene and by the thickness of the horny layer. The amount of melanin produced is influenced by genetic factors, the amount and wave lengths of UV light received the amount of melanocyte. Stimulating hormone (MSH) secreted, and the effected of melanocyte stimulating chemical such as furocoumarins (psoralens) Abnormal pigmentation of the skin is produced by a variety of causes.

**Types of pigmentary disorder:**

Excessive pigmentation is known as hyper pigmentation and decreased pigmentation is known as hypo pigmentation. Both may be localized or generalized. In addition, increased pigmentation may result from deposits of

abnormal non melanin pigments in the skin, Eg, Haemosiderin from broken down haem pigment in extravasated blood.

Homogentisic acid deposited in cartilage particular in the inherited metabolic defect known as alkaptonuria.

Drugs and heavy metals toxicity. Silver, Gold, Mercury, Arsenic poisoning, Amiodarone and phenothiazines causes slate grey, dusky skin pigmentation in exposed sites.

### **Vitiligo:**

The name 'Vitiligo' is derived from the Latin word skin eruption, victim meaning a blemish (spoil the beauty of) happens to be a synonym for it. White skin is the literal meaning of leucoderma, derma being derived from the Greek words, leucas and dermis. Leucas means white and dermis means skin.

Celsus was the first Roman physician of the 2<sup>nd</sup> century to coin the word vitiligo, because the disease resembles the white patches of a spotted calf (vitellus).

Being a social problem. WHO announced May 19<sup>th</sup> as “**World Vitiligo day**” to create awareness.

### **Prevalence:**

About 0.5 to 1 percent of the world's populations, or as many as 65 million people, have vitiligo. In the United States, 1 to 2 million people have the disorder. Half the people who have vitiligo develop it before age 20; most develop it before their 40<sup>th</sup> birthday. The disorder affects both sexes and all races equally; however, it is more noticeable in people with dark skin.

**Epidemiology:**

Vitiligo is an acquired idiopathic depigmentary condition which, though worldwide in distribution, is most common in India. Egypt and other tropical countries. It is a source of great social embarrassment of dark skinned people. It affects all age groups with no predilection to either sex.

**Gross Anatomical changes in Vitiligo:**

Vitiligo represents an acquired patchy loss of pigments of the skin. There are no gross changes seen except irregularly demarcated. Hypo pigmented patches of varying size, usually surrounded by hyper pigmented skin. These are seen distribute symmetrically or asymmetrically at various parts of the body.

**1. Definition:**

Vitiligo is an acquired often disfiguring, pigmentary anomaly of the skin manifested by depigmented white patches surrounded by a normal or a hyperpigmented border.

As a result, white patches appear on the skin in different parts of the body. similar patches also appear on both the mucous membranes ( tissues that line the inside of the month and nose), and the retina (inner layer of the eyeball). The hair that grows on areas affected by vitiligo sometimes turns white.

It is an extremely common depigmentary disorder of great medicosocial significance among the dark people, etiology is uncertain association with variable penetrance; a symptomatic puncture linear, oval, circular or irregular, discrete or confluent depigmented and or hypopigmented macules on otherwise normal skin is confined to mucocutaneous functions dermatomal unilateral or bilateral, symmetrical or asymmetrical generalized or universal over laying hair retain pigment or turn white, no autonomic or sensory disturbances, sun burn or chronic solar damage in longstanding cases, unpredictable and capricious course, stationary, self healing or progressive.

It is quite clear that vitiligo is due to some derangement in the pigment metabolism resulting in appearance of white patches in the skin. It is hard to say whether the site of derangement is usually general or local, but the main affected part is the skin, which is the most exposed part of the body. It can be examined by naked eye and can furnish a lot of information about the person and the disease. In certain cases the changes are not clear. Hence the study of the skin structure and its physiology is essential for proper assessment.

## **II. Etiology & Pathogenesis:**

- Epidemiological studies suggest that vitiligo or a susceptibility to the disease may be inherited and about one fourth to one third of patients have family members affected with the disease. A multifactorial pattern of inheritance is revealed in most studies.
- Three possible mechanisms that may cause destruction of melanocytes, the pigment – producing cells of the skin, have been suggested by different workers.
- The **autoimmune hypothesis** originated from the observation that vitiligo is associated with some autoimmune diseases. Both cellular and humoral factors responsible for autoimmune damage to melanocytes have been demonstrated.

These autoimmune diseases include hyperthyroidism (an overactive thyroid gland), adrenocortical insufficiency (the adrenal gland does not produce enough of the hormone called corticosteroid), alopecia areata (patches of baldness), and pernicious anemia (a low level of red blood cells caused by the failure of the body to absorb vitamin B<sub>12</sub>), and it is also common in diabetes mellitus.

Scientists do not know the reason for the association between vitiligo and these autoimmune diseases. However, most people with vitiligo have no other autoimmune disease.

- The **autocytotoxic or self – destruct hypothesis** suggests that some toxic molecules produced during the biosynthesis of melanin are responsible for melanocyte damage in susceptible individuals.
- The **neural hypothesis** postulates that neurochemicals liberated from nerve endings are toxic to melanocytes.

Drugs and chemicals – like quinines, guano furacin, amyphenol, chlorthiazide, broad spectrum antibiotics and chloroquin.

It is also regarded to develop through eczema scar of prick by injection needle, injury by burn or from other accidents, by friction of foot, wearing tight clothes. It has also been observed in persons who have suffered serious illness due to typhoid, jaundice, liver diseases, diabetes, worms, constipation and diarrhea.

The non pigmented patches whitish or reddish are round or oval in shape with smooth surface and slowly grow into large, irregularly outline areas. It may be the result of skin disease or it may be harmless conditions of unknown cause.

- **Hereditary factors**

Hereditary is one of the factors supposed to be related with vitiligo. Familial incidence has been reported in 7.5 to 21% in India and 33 to 40% in Western countries.

- **Emotional factors**

It is every day knowledge and observation that emotional factors affect the skin as shown by the blushing of embarrassment, the pallor of fear and the pallor or redness of change, depending on the subject and his emotional state. Experiments have demonstrated that emotional change can affect the following, which has direct relevance in the etiology of certain skin disorders.

- Control of vascularity of the skin
- Control of sebaceous gland secretion
- Influencing the degree of oxidation.
- Influencing the tendency of pruritus.

There is due to the causative factor of this disease, venpapai from the following basic facts, it is generally considered to be a trophoneurosis. Psychological factors are known to be responsible for the precipitation and aggravation of the disease.

### • Others

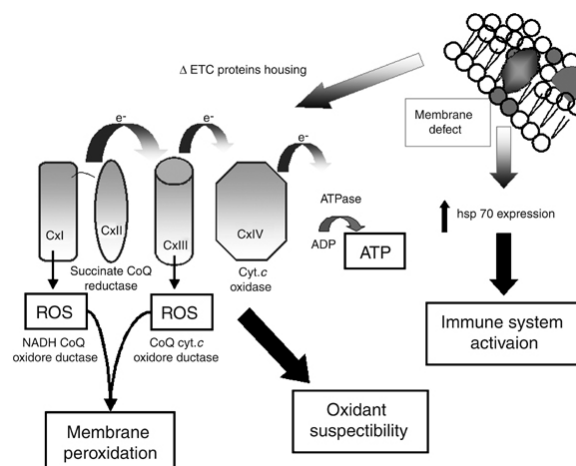
Sometimes vitiligo can be caused by the action of monobenzyle either on hydroquinone presents in the slippers, gloves (or) other articles made of rubber or used as a depigmenting agent in the form of an irritant for pigmentary disorders. Recently vitiligo has also been observed to occur from plastic slippers as well as plastic 'hindis'.

### III Pathology:

Chemically melanin pigment is a group of chromo proteins with coloured prosthetic groups, which is derived from the precursor tyrosine in the following way Tyrosine tyrinase dihydroxy phenylanin (DOPA) Melanogenase Melanin (Dopa Oxidase).

Melanin + Protein = Melano protein

The possible pathogenetic mechanism, a schematic picture of the causal and temporal sequence leading to the melanocyte functional impairment during vitiligo.



In the skin, the pigment is produced by the melanocytes of their precursor's melanoblasts. The melanoblasts are supposed to be derived from the cells of neuro ectodermal origin during the embryonic life. After birth, these cells migrate to their definitive position. The melanocytes appear as clear cells within the basal cell layer of the epidermis and show dendritic processes after special staining. These processes come in contact with similar process of other melanocytes and epithelial cells through which the melanin pigments are donated to the basal cells of epidermis. The dermis of normal skin also shows macrophages containing melanin pigments known as melanophores, which are incapable to produce the melanin pigments.

#### **Histopathologic changes in vitiligo:**

Marked histological changes do not occur in cases of Vitiligo. All the layers of the epidermis and dermis appear normal except a few changes which can be seen after special stains.

In the affected area the basal cells and the keratinizing cells of the other layers of epidermis do not contain melanin pigment granules in them. The contrast can be seen at a junction of the normal and vitiliginous areas of the skin, especially by silver staining or DOPA reaction. The pigment cells, the melanocytes are not seen in the affected area but they are present in the adjacent normal skin. At the border of the patches of vitiligo the melanocytes often appear large and possess long dendritic process filled with melanin granules. Electron microscopic studies confirm the absence of melanocytes in areas of long standing vitiligo.

There are collections of mononuclear cells at dermo epidermal junction at the border between vitiliginous and normal skin. These cells are predominately small lymphocytes. In the long standing case where the skin has become thick and scaly, varying amount of the keratosis is seen.

#### **IV. Clinical Features:**

- Vitiligo affects all races with an average frequency of 1 to 2 percent of the population. Both sexes are affected equally and the disease may develop at any age. The peak age of onset in most series was between 10 and 30 years. Stressful life events or physical trauma can often precipitate the onset of disease.
- The typical macule of vitiligo is easily recognized by well circumscribed milky white spots of varying sizes without any other discernable surface change of the skin. The hairs on the patch may turn gray or white (Leucotrichia). There may be a single spot or numerous white macules distributed all over the body. With passage of time, the macules may enlarge and coalesce to produce extensive pigment loss. The lesions are symptomless.
- Occasionally the depigmented areas are slightly pink at the start of the disorder.
- Often the depigmented patches are symmetrical, especially when the disorder is distributed over the peripheral parts of the limbs and the face.
- These patches are more commonly found on sun-exposed areas of the body, including the hands, feet, arms, face, and lips. Other common areas for white patches to appear are the armpits and groin, and around the mouth, eyes, nostrils, navel, genitals, and rectal areas.
- In addition to white patches on the skin, people with vitiligo may have premature graying of the scalp hair, eyelashes, eyebrows, and beard. People with dark skin may notice a loss of colour inside the mouths.
- Odd patterns are sometimes noted as for example, when the depigmentation occurs over the front of the neck over the thyroid gland, or on the abdomen over the site of the pancreas or on the flanks over the sites of the adrenal glands.



- Vitiligo lesions may result from ‘Koebner phenomenon’ i.e. appearance of new lesions at sites of non-specific trauma such as abrasion, surgical scars, severe sunburn or inflammatory skin diseases like psoriasis or eczema.
- Vitiligo is most noticeable in the summer when the normal skin is tanned by the sun.
- Vitiligo sometimes disappears spontaneously after months or years but more usually the conditions spreads slowly and may eventually involve nearly whole of the skin.
- Early lesions may be pale white and ill defined. At this stage, Wood’s lamp helps to confirm the diagnosis. Patches enlarge slowly and may affect the whole body.

## V. Types:

- \* **According to the extent of involvement** and pattern of distribution, vitiligo is clinically categorized into focal, segmental, generalized, acrofacial, and universal types.
- \* **Focal Vitiligo** is and isolated macule or a few macules in a localized non-dermatomal distribution.
- \* **Segmental vitiligo** is characterized by macules in a unilateral dermatomal distribution. This type of disease usually pursues a stable course.
- \* **Generalised vitiligo** is the most common type showing macules in a generalized widespread distribution. There is often striking symmetry of affection and involvement of extensor surfaces. Face (particularly around the orifices), neck, bony prominences of hands, legs: axilla and mucosal surfaces are particularly affected.
- \* **Acrofacial vitiligo** affects distal end of fingers and facial orificies in circumferential pattern.

- \* **Universal vitiligo** implies loss of pigment over the entire body surface areith only isolated islands of normal pigmentation remaining.

## **VI Associated diseases:**

- \* Patients with vitiligo have in increased risk of developing autoimmune diseases like thyroid diseases, Addison's disease, pernicious anemia and insulin-dependent diabetes mellitus. Auto antibodies against other organs may be detected in the absence of clinical evidence of the disease. Premature graying of hair and alopecia areata are important cutaneous associations in some patients.
- \* The pigment epithelium of retina and choroid are developmentally derived from the neural crest, the cutaneous melanocytes originate from the same embryonic structure. They ma share the susceptibility to damage in vitiligo; iris and retinal pigmentary anomalies in the absence of ophthalmologic complaints may be detected in a proportion of the patients. Iris may be found in a small number of patients.

## **Psychosocial Impact of Vitiligo**

- \* Although vitiligo by itself is symptomatic and does not cause any physical discomfort or disability, it may be associated with devastating psychological and social consequences. Since a person's appearance is a major determinant of his/her personality traits, vitiligo, by causing cosmetic blemishes can have major impact on personality.
- \* Feeling of stress and embarrassment on social contacts, lack of confidence and lowered self esteem may be detrimental to the patients, particularly when the spots are a visible area of the body.

- \* The psychological impact can have serious implications in deeply pigmented races such as Indians, in whom the contrast between the normally dark skin and the white lesions can be marked.

### **Clinical Criteria for Classification of Vitiligo:**

#### **Stages of Clinical Features:**

#### **Vitiligo**

<b>Active (V1)</b>	(i)	New lesions developing
	(ii)	Lesions increasing in size.
	(iii)	Border ill-defined
<b>Quiescent (V2)</b>	(i)	No new lesions developing
	(ii)	Lesions stationary in size
	(iii)	Border hyper pigmented and well defined.
<b>Improving (V3)</b>	(i)	Lesions decreasing in size
	(ii)	No new lesions developing
	(iii)	Border defined and signs of spontaneous repigmentation

**Zosteriform:** Unilateral distribution of lesions, preferably along the course of nerves. Besides typing the stage of disease, it is useful to decide the variety (acral, vulgaris, zosteriform), Severity (Localized or extensive) and acuity (insidious or galloping) or vitiligo.

### **VII Diagnosis:**

- The diagnosis of vitiligo is made based on a physical examination medical history, and laboratory tests.
- **Physical examination:** White patches of skin on the body particularly on sun-exposed areas, including the hands, feet, arms, face and lips.
- **Medical History:** Important factors in the diagnosis include a family history of vitiligo; a rash, sunburn, or other skin trauma at the site of vitiligo 2 to 3 months before depigmentation started; stress or physical

illness; and premature (before age 35) graying of the hair and ask whether patient or anyone his family has had any autoimmune disease, and whether patients are very sensitive to the sun.

- **Laboratory tests:** To help confirm the diagnosis, we make take a small sample (biopsy) of the affected skin to examine under a microscope. In vitiligo, the skin sample will usually show a complete absence of pigment-producing melanocytes. On the other hand, the presence of inflamed cells in the sample may suggest that another condition is responsible for the loss of pigmentation.
- It is usually apparent; in doubtful and early case. Wood's lamp is of great help in diagnosis.
- These areas often fluorescence a golden yellow when examined under a Wood's lamp. The hypomelanotic macules in leprosy are anesthetic.
- Examination of the skin in long wave UVR helps distinguish whether there is total depigmentation (as in vitiligo) or not. it may also detect areas of depigmentation not easily seen in ordinary daylight, as well as detecting a lemon-yellow fluorescence seen in some cases of pityriasis versicolour.

### **VIII Course and prognosis:**

- The common generalized vitiligo usually pursues a course of slow progression with enlargement of existing macules and gradual appearance of fresh spots. Quite often, after an initial phase of progression, the lesions remain relatively stable for varying periods of time only to be followed by accelerated spread, sometimes there may be very rapid spread leading to extensive loss of pigmentation within a short span of time. In an individual case the course however is unpredictable.
- In comparison with the aforesaid, segmental vitiligo tends to have a very stable course. Following appearance of lesions in a dermatomal distribution, the lesions usually remain localized to the area of affection.

- Spontaneous repigmentation may be observed in a proportion of patients particularly in lesions on sun-exposed areas. However, the extent of spontaneous healing is seldom cosmetically significant.

It has improved considerably in recent years because of better understanding of etiological factors and advances made in therapy. Following conditions are said to be of poor prognosis.

- (i) Poor nutritional state or digestion, use of broad – spectrum antibiotics over long period. Emotional stress and nervous debility.
- (ii) Presence of vitiligo on resistant sites like the hands and the feet, front of wrists, the elbow, the waist, the eyelids and lips.
- (iii) Depigmented hair in vitiliginous areas.

#### **Other Causes of Hypo Pigmentation:**

**Generalised depigmentation** is found mostly in albinos. In this case, the characteristic dendritic melanocytes are present in the skin, but they are unable to produce melanin pigment due to defective tyrosinase activity. In albinism, the skin looks milky white, the hairs are pale looking and the iris is transparent. The generalized pallor is also noticed in panhypopituitarism, male eunuchoidism and phenyl ketouria.

**Located depigmentation** is often noticed in the skin of pattern leucoderma. The white patches on the skin may be quite extensive and the condition is inherited as an autosomal dominant character.

Sometimes sharply defined focal depigmented areas are found on skin of persons suffering from vitiligo. In the affected areas, melanocytes are absent and there is no trace of melanin. The condition is an acquired one and shows some familial tendency.

**Leucoderma:**

Leucoderma may be defined as a type of acquired skin depigmentation produced by some specific substances (or) dermatosis several types of Leucoderma may be seen.

1. Occupational Leucoderma may occur in those who work in rubber garments (or) wear gloves that contain antioxidant monobenzyl ether of hydroquinone many phenolic compounds can produce Leucoderma.
2. Postinflammatory Leucoderma may result from many inflammatory dermatoses such as, Pityriasis rosea, psoriasis, herpes zoster, secondary syphilis, and morphea.

Leucoderma is also commonly seen on the flanks of ladies wearing tight petticoat strings where the prolonged pressure is presumed to lead to depigmentation.

**Piebaldism:**

In this condition there is a white forelock and white patches on the skin surface.

In Waardenburg's syndrome the condition associated with sensory deafness.

Pityriasis Versicolour	Superficial fungus infection leading to disturbance in pigment production, common multiple pale scaling patches on trunk.
Pityriasis alba	Mild patchy eczema of the face in children causing a disturbance in pigment production.
Leprosy	One or several paler macules on trunk or limbs that are hypo aesthetic.
White macules of affecting tuberous sclerosis	Uncoming development of anomaly of CNS, connective tissue and skin; several 'maple leaf' shaped hypopigmented macules.
Postinflammatory hypopigmentation	After inflammatory skin disease (after eczema or trauma to the skin, irregular in shape and in depth of pallor).
Naevous annemicus	Rare developmental solitary white patch usually on trunk; thought to have vascular basis.
Chemical toxicity	May look very much like vitiligo; seen in workers in rubber industry exposed to parateriary benzyltoluence.

## DIFFERENTIAL DIAGNOSIS OF THE IMPORTANT DEPIGMENTARY DISORDERS.

Distinguish features	Albinism	Naevus dpigmentosus	Vitiligo	Leprosy	Pityriasis
Age	Congenital present at birth	Congenital present at birth	Acquired	Any age	Any age
Distribution	Complete (or) Partial	Unilateral	Any area	Any area	Trunk, Neck, and Face.
Course	Stationary	Does not increase in size or changing shape	Progressive	Progressive	Progressive worse in monsoon and summer
Hyperpigmentary border	Nil	Nil	Present	Inflammatory	Nil
Heredofamilial	Hereditary	Not hereditary	Rare	Nil	Nil
Other features	Hair and eyes may be affected	Nil	Hair may be affected	Anesthesia thickened nerves, nasal, bleeding slit smear and biopsy	Furfuraceous like dandruff, scaling in head macules and large patches.

# TRIAL DRUGS

## LITERATURE REVIEW OF TRIAL DRUGS

### 1) ótuR - Puvarasu

Botanical name : Thespesia populnea.Linn.soland  
 Family : Malvaceae  
 ntW bga® : òéuhr, ód«  
 Rit : if¥ò, Jt®¥ò  
 j<ik : bt¥g«  
 ãçî : fh®¥ò  
 brœif : òGibfhšè  
Jhœikahj»

Fz« :

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-Fzghl« Kjš ghf« gjf« v© 705

- F£I«, foNiy, élghf«, knfhju«, nrhig,fu¥gh<, »uªÂ,nkf« nghF«.

### 2. Ezh

Botanical name : Morinda tinctoria



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jzᳵF, EzĬ  
Rit : fh®¥ò  
jᳵik : bt¥g«  
ÃĈĬ : fh®¥ò  
brœif : ÅᳵfKUᳵ»  
ĭiy,fhœ : UJĬ©lhᳵ»  
gH« : cuK©lhᳵ»  
bt¥gf%œĭ  
bt¥gK©lhᳵ»  
nt® : bgUky«nghᳵ»  
bghJFz« :

gɛil fu¥gbdhL ghuĉÁ nyZkRu«

xɛo ãĭ ò©»uᳵÂ xɛLšfh©-kɛlyiu

VᳵJ Ezhéᳵ ĭiykᳵj« Ô®ᳵJešy

fhᳵÂjU nkfkLš fh©

-Fz¥ghl« Kjš ghf« - gᳵf« v© 590

fu¥ghᳵ, laRu«, ò©, fhᳵiy, khᳵj« ,nkf« ĭitfis xêᳵF« , clš xëiaᳵ jU«

### 3. ešby©zœ (vŸëᳵ beœ)

Gingelly oil

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clš óç¥ò, clš t«ik M»at%.iw«

jUtnjhL,f©nzhŒ,fhJnehŒ, jiy<sub>i</sub>bfhÂ¥ò

brhç, Áu§F, ò© Kjèaitfisí« ngh<sub>i</sub>F«.

## TRIAL MEDICINES

1. ótur« g£il FoÚ® Nuz« :-

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x«wh£ t%¿a Ã« tof£o vL»Jî bfšYšš

msî 60 ml ĨUntis

2. Ezh ijy«:-

br£Kiw

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bfšYšî«.

Ãunahf«:- btëÃunahf«

ÔU«neh£:- bt©òYë Ú§F«.

--Fzgħl« Kjš ghf« gîf« v© 591

## **MATERIALS AND METHODS**

The clinical study of Swatha Kuttam was carried out in post graduate department of Maruthuvam, Govt. Siddha Medical College, attached to Arignar Anna Hospital, Arumbakkam, Chennai – 600 106.

### **Selection of Cases:**

The population consists of all patients with Swatha Kuttam (completely depigmented or hypopigmented patches without any structural change in the skin) satisfying the inclusion and exclusion criteria mentioned below.

### **Sample Size:**

The trial size will be 40 patients. (Both Male and Female)

### **Data Collections:**

Literary evidence collected from various

- Siddha Literature
- Books on Modern medicines
- Medical Journals
- Internet

### **Inclusion Criteria:**

1. Age between 10 to 55 years.
2. Willing to give specimen of blood for investigation when required.
3. Willing to attend OPD once in 15 days for 3 months.
4. Hypo-pigmentary patches
5. Patient having white or pink coloured patches

**Exclusion Criteria:**

1. Patients of Jaundice
2. Hypopigmented patches of leprosy and burns
3. Connective tissue disorders.
4. Heart ailments are not eligible for this trial
5. Fungal infection
6. Worm infestation

**Withdrawal Criteria:**

1. Any drastic changes occurring in hematological findings during treatment period.
2. Development of any exacerbation in clinical features.

**Trial Period:**

- 3 Months

**Evaluation of Clinical Parameters:****Clinical Assessment**

Site  
Size  
Colour  
Margin  
Shape  
Itching.

**Investigations:**

Blood Test : RBC, Hb, Tc, Dc, ESR, Blood Sugar  
Serum Cholesterol, Blood Urea.  
Urine Test : Albumin, Sugar, Deposit, Serum Creatinine  
Motion Test : Ova, Cyst

**Siddha Aspects:**

**1. Envagai Thervu**

Naa

Niram

Mozhi

Vizhi

Sparism

Naadi

Malam

Moothiram

**2. Neerkuri**

Niram

Manam

Nurai

Enjal

Eadai

**3. Neikuri**

**Conduction of the study**

Swatha Kuttam patients satisfying the inclusion and exclusion criteria will be included in the trial. Informed consent will be obtained from the patients. A day before starting the trial treatment, cleaning of mukkutram by purgation will be carried out by Agasthiyar Kuzhambu, early morning 100mg with hot water.

For O.P. patients, the trial drugs will be issued for 7 days at a time. They will be asked to attend the O.P. department once in 7 days.

**Case Sheet Proforma:**

All clinical signs and symptoms of “Swatha Kuttam” history of the present and the past illness, personal history, family history, habits and occupation were recorded. Lab investigation and prognosis were recorded for analysis.

**Trial Drug, Dosage:**

The following medicine were selected on the basis of Siddha Literature and given.

1. Puvarasam Pattai Kudineer Choornam 25 gm – 60ml twice a day - Internal
2. Nuna Thylam - External

## **RESULTS AND OBSERVATION**

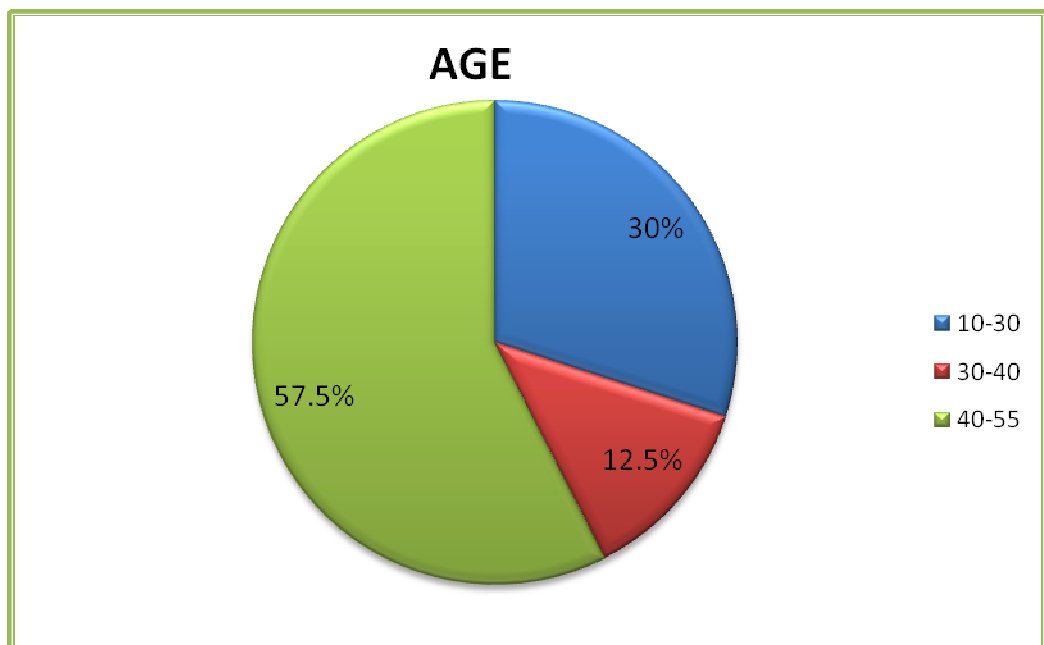
A Total number of 40 patients were treated in O.P of PG Maruthuvam Department attached with Arignar Anna Govt., Hospital of Indian Medicine during the period 2010-2012. All patients included in the study with signs and symptoms of Swetha kuttam were observed. The observations were tabulated regarding the following features.

- AGE REFERENCE
- RELIGION REFERENCE
- SEX REFERENCE
- SOCIO ECONOMIC STATUS
- ETIOLOGY REFERENCE
- DIET REFERENCE
- FAMILY HISTORY REFERENCE
- THINAI
- SITE OF LESION
- PARUVA KAALAM
- DERANGEMENT IN THE TYPES OF VAATHAM, PITHAM, KABHAM
- UDAL KATTUGAL
- ENVAGAI THERVU
- DISTRIBUTION OF NAADI AMONG THE PATIENT WITH SWETHA KUTTAM
- NEIKURI REFERENCE
- CLINICAL FEATURES BEFORE & AFTER TREATMENT
- GRADATION OF RESULTS



## AGE REFERENCE

Age (In Years)	No. of Cases 40	Percentage %
10-30	12	30.0
30-40	5	12.5
40-55	23	57.5

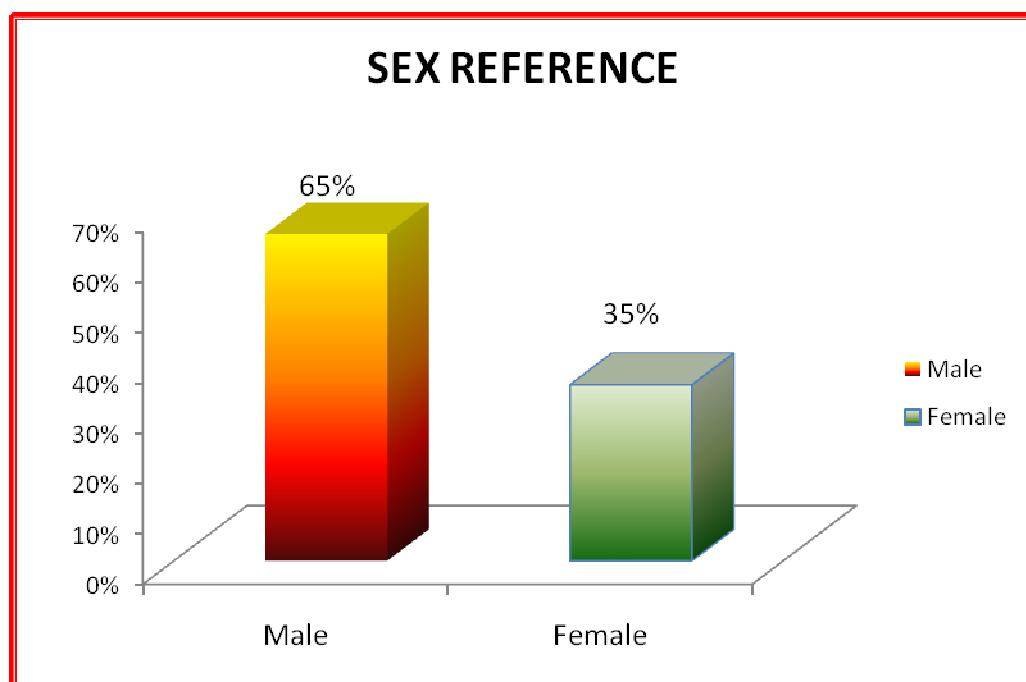


## Inference

About 57.5% of them were aged 40-55 years and 12.5% cases 30-40 years, 30% cases 10-30 years.

## SEX REFERENCE

Sex	No. of Cases Out of 40	Percentage %
Male	26	65
Female	14	35

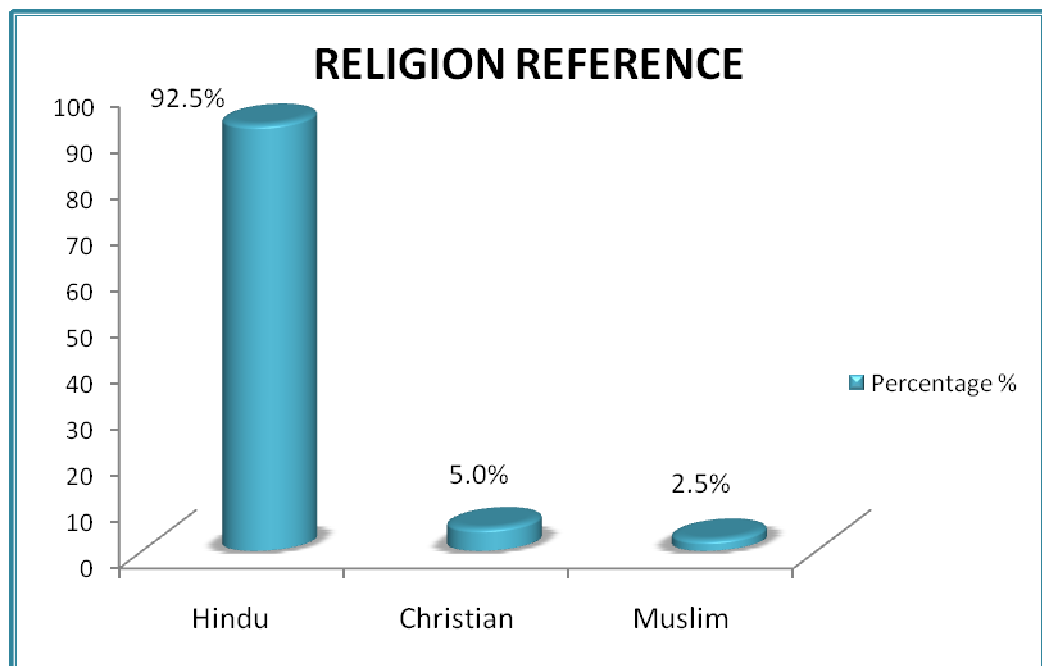


## Inference

65% were Male cases and 35% were Female cases.

## RELIGION REFERENCE

Religion	No.of Cases Out of 40	Percentage %
Hindu	37	92.5
Christian	2	5
Muslim	1	2.5

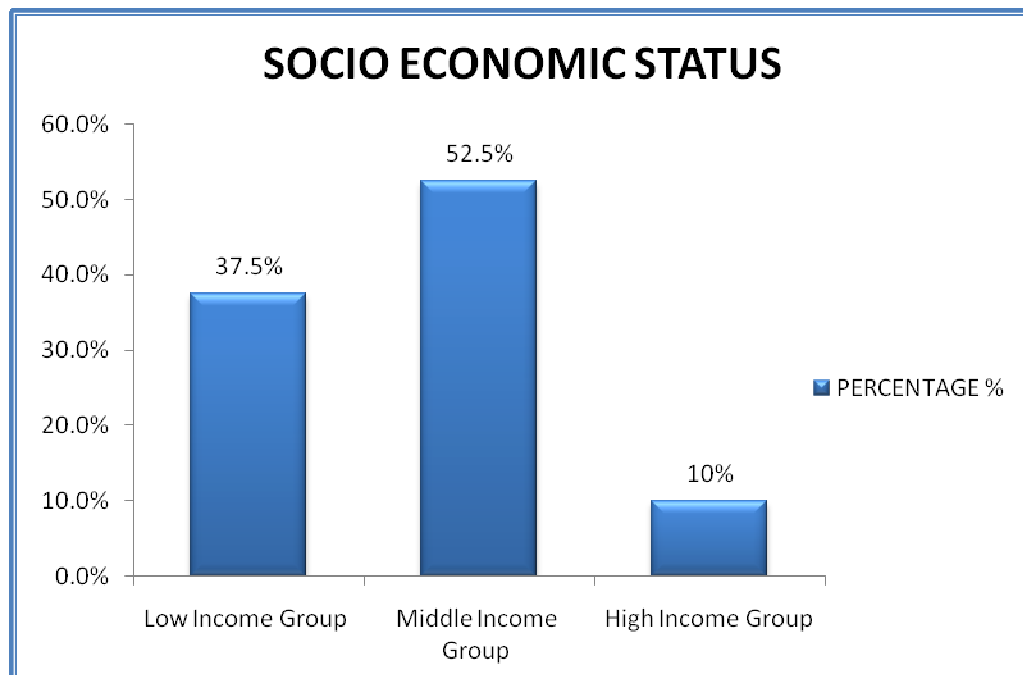


### Inference

Most of the patient's religion Hindu 92.5%, Christian 5% and Muslims 2.5%

## SOCIO ECONOMIC STATUS

Socio Economic Status	No.of Cases Out of 40	Percentage%
Low Income Group (Below Rs.10000)	15	37.5%
Middle Income Group (Rs.10001 – Rs.20000)	21	52.5%
High Income Group (Above Rs.20000)	4	10%



### Inference

About 52.5% of the patients were middle income group, 37.5% lower and 10% high income group.

## ETIOLOGY REFERENCE

<b>Etiology</b>	<b>No.of Cases Out of 40</b>	<b>Percentage %</b>
Hereditary	3	7.5%
Autoimmune diseases	4	10%
Unknown	33	82.5%
Worm Infestation	0	0

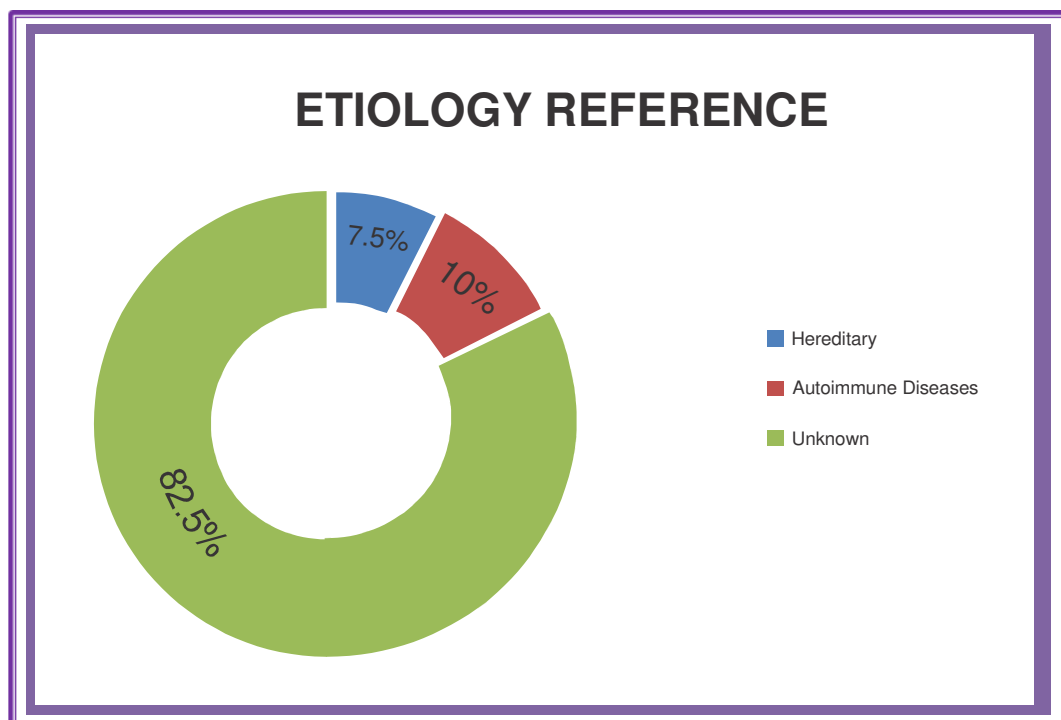


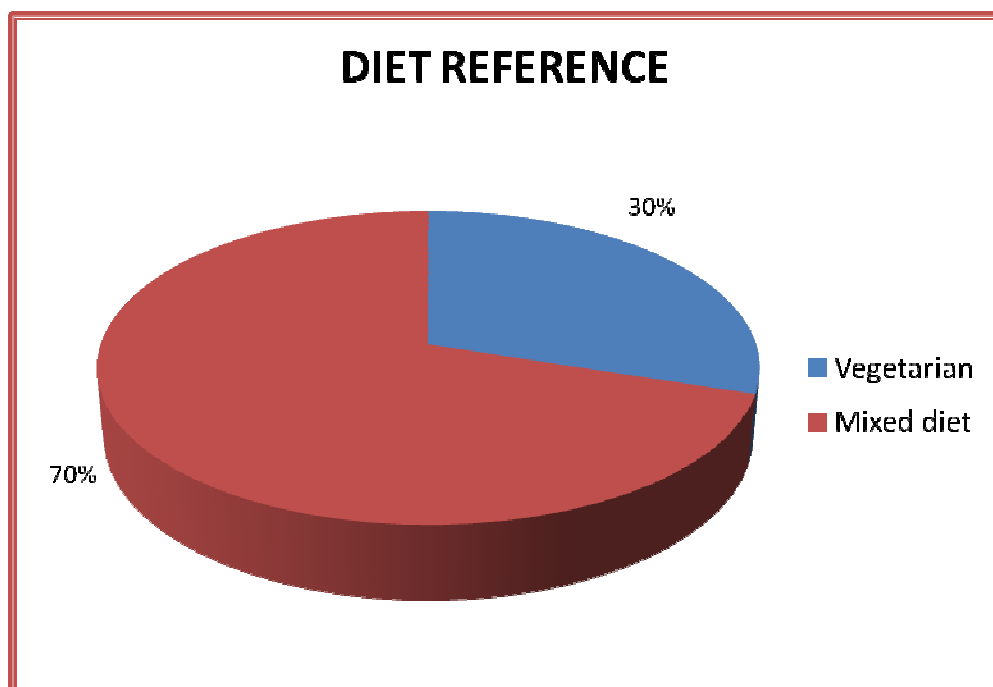
Figure 1

### Inference

About 50% of the cases were 7.5% Hereditary, 10% Autoimmune diseases, 82.5% Unknown Etiology.

## DIET REFERENCE

Food Habits	No.of cases out of 40	Percentage %
Vegetarian	12	30%
Mixed Diet	28	70%



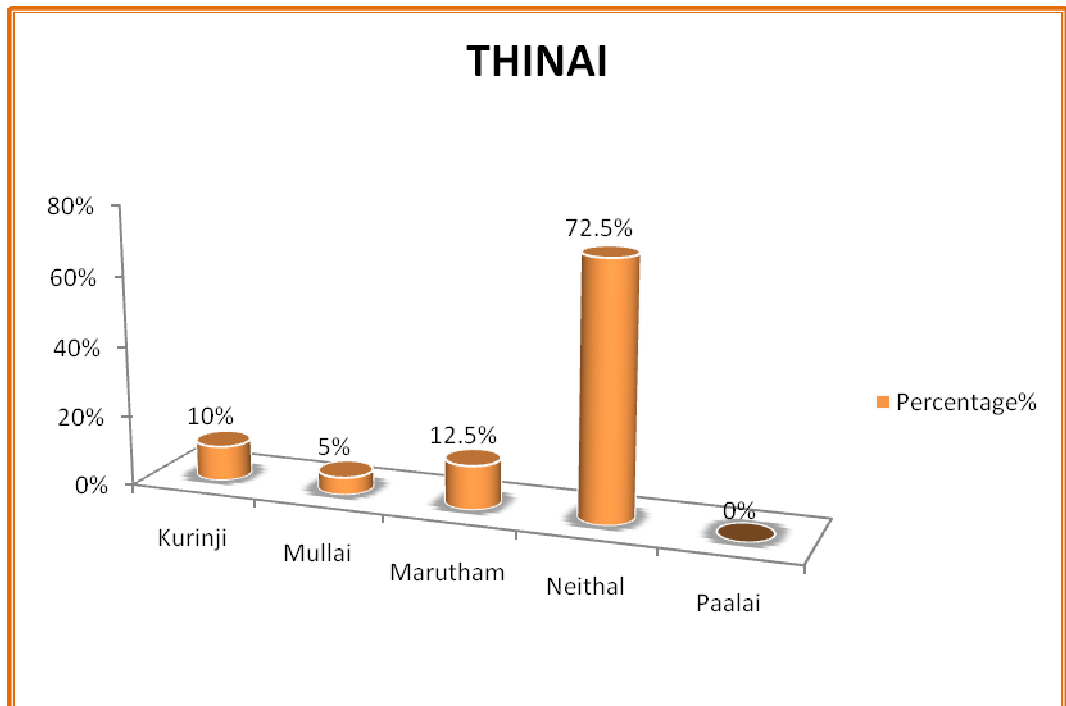
### Inference

70% of cases were taken mixed diet.

30% of cases were taken Vegetarian.

## THINAI

Thinai	No.of Cases Out of 40	Percentage%
Kurinji	4	10%
Mullai	2	5%
Marutham	5	12.5%
Neithal	29	72.5%
Paalai	0	0%

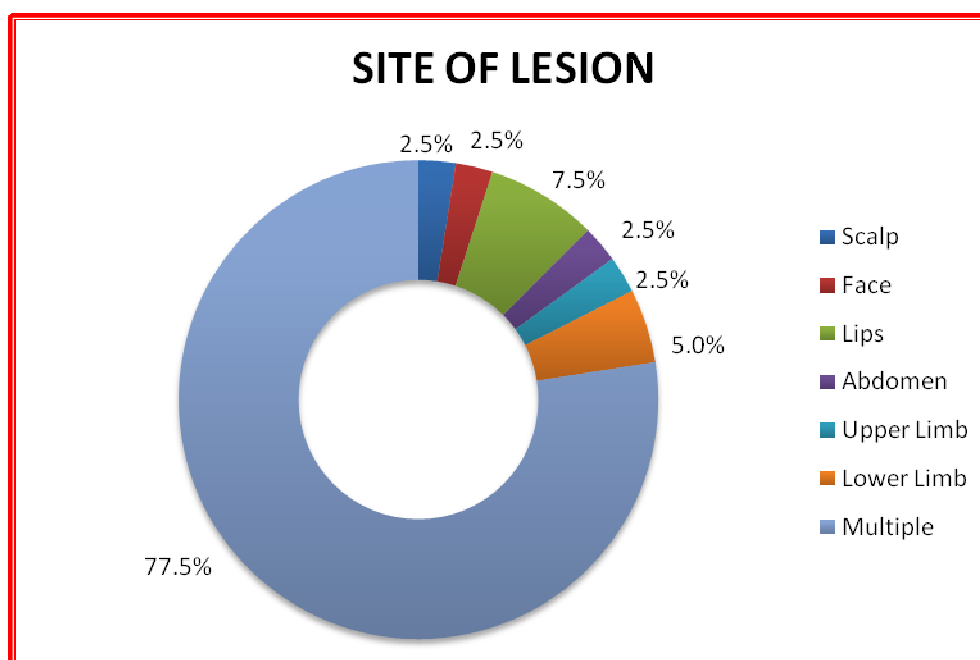


## Inference

According to Thinai the highest distribution 72.5% was noted in Neithal, in Marutham 12.5% and Kurinji 10% and Mullai 5% were observed.

## SITE OF LESION

Site of Lesion	No.of cases out of 40	Percentage %
Scalp	1	2.5%
Face	1	2.5%
Lips	3	7.5%
Abdomen	1	2.5%
Upper Limb	1	2.5%
Lower Limb	2	5.0%
Multiple	31	77.5%



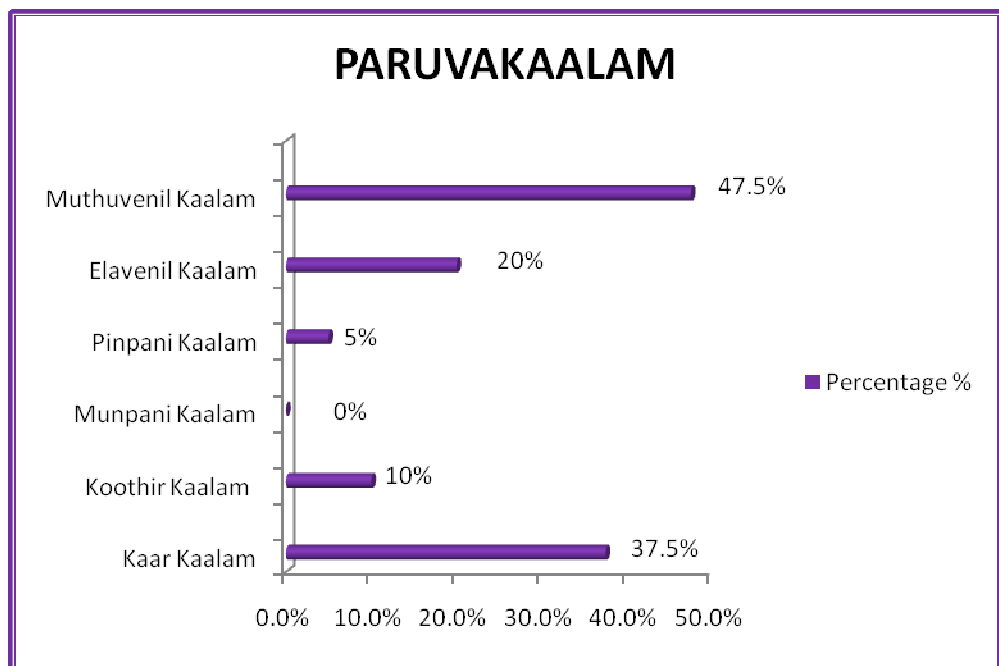
## Inference

About 77.5% of cases were having multiple site of lesion. Lower limb 7.5% Scalp region 5%, lips 2.5%, Abdomen 2.5%, upper limb 2.5%, face 2.5%.



## PARUVA KAALAM

Paruvakaalam	Months	No.of cases out of 40	Percentage %
Kaar Kaalam	Avani- Purattasi (Mid Aug-Mid Oct)	15	37.5%
Koothir Kaalam	Ippasi,-Karthigai (Mid Oct – Mid Dec)	4	10%
Munpani Kaalam	Margazhi,-Thai (Mid Dec – Mid Feb)	0	0%
Pinpani Kaalam	Maasi,-Panguni (Mid Feb – Mid april)	2	5%
Elavenil Kaalam	Chithirai,-Vaigasi (Mid April – Mid June)	8	20%
Muthuvenil Kaalam	Aavi- Aadi (Mid June – Mid Aug)	19	47.5%

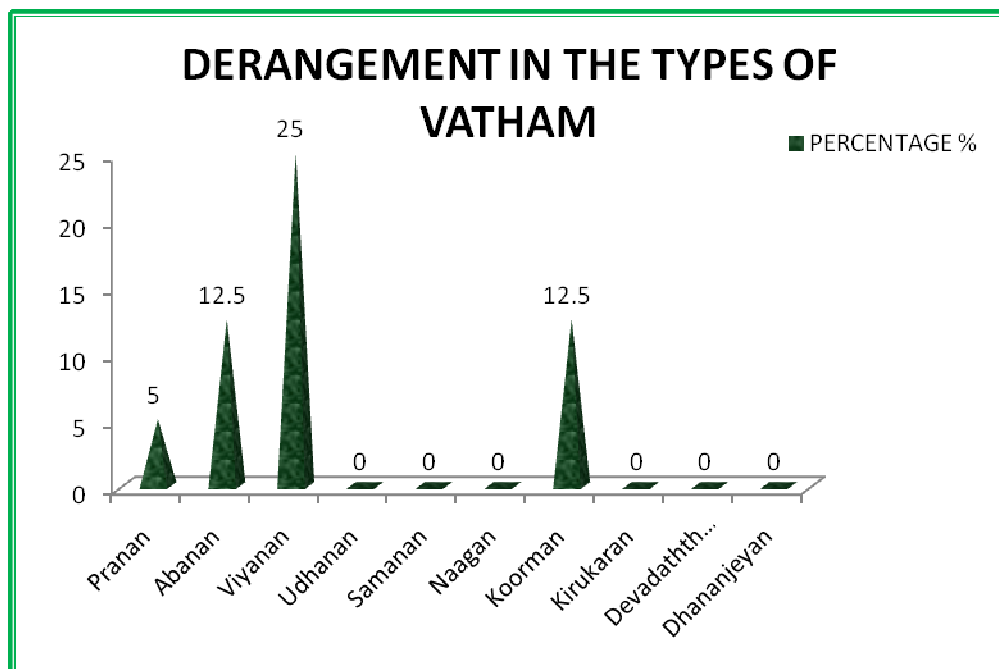


## Inference

According to paruvakaalam highest incidence 47.5% were noted in Muthuvenil kaalam 37.5% cases were noted in Kaarkaalam, 20% were noted in Elavenil kaalam 10% were noted in Koothir kaalam. Only 5% were noted in Pinpani kaalam.

## DERANGEMENT IN THE TYPES OF VATHAM

Vatham	No.of Case Out of 40	Percentage %
Praanan	2	5%
Abaanan	5	12.5%
Viyanan	10	25%
Uthanan	0	0%
Samanan	0	0%
Nagan	0	0%
Koorman	5	12.5%
Kirugaran	0	0%
Devathanan	0	0%
Dhananjeyan	0	0%

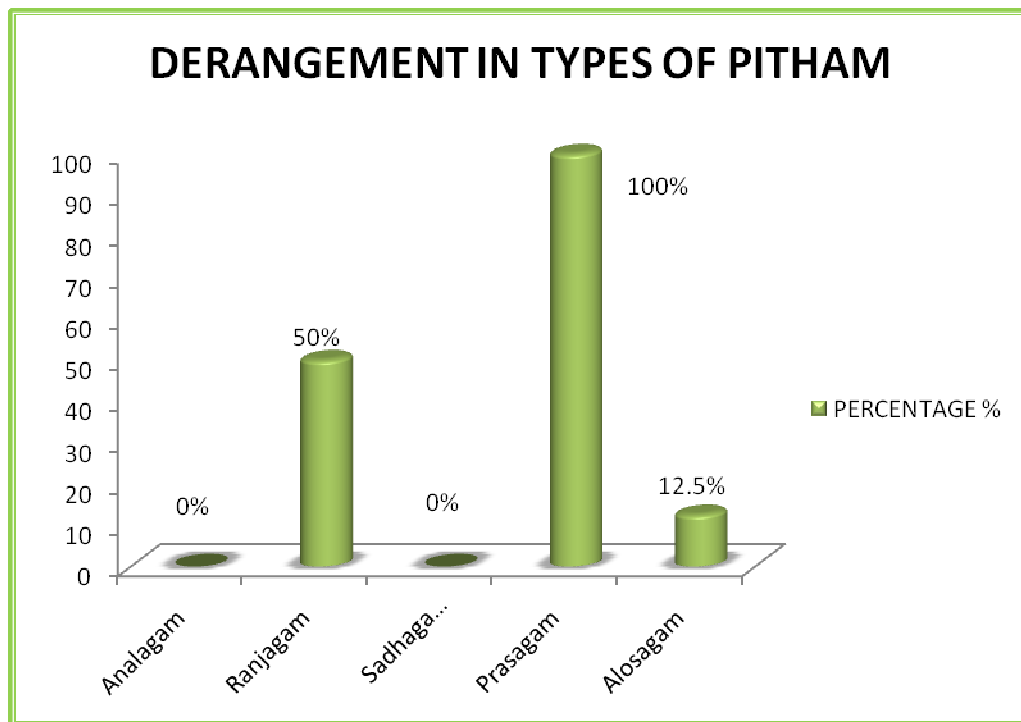


### Inference

According to classification of Vatham, Abanan and Koorman were deranged in 12.5% of cases, Piranan was deranged 5%, Viyanan was deranged in 25% of cases.

## DERANGEMENT IN TYPES OF PITHAM

Pitham	No.of Cases Out of 40	Percentage %
Anar pitham	0	0%
Ranjagam	20	50%
Saathagam	0	0%
Prasagam	40	100%
Alosagam	5	12.5%

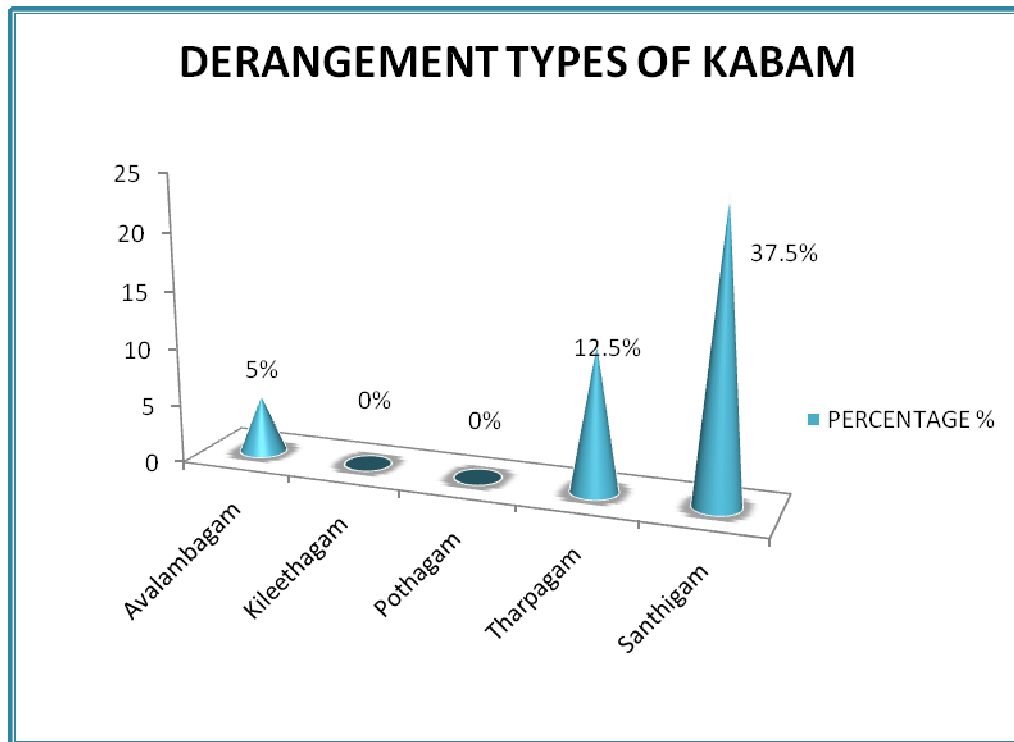


### Inference

According to Pitham Prasagam was deranged in 100% of cases, Ranjagam was deranged in 50%, Alosagam was deranged 12.5%.

## DERANGEMENT TYPES OF KABAM

Types of kabam	No.of Case out of 40	Percentage %
Avalambagam	2	5%
Kilethagam	0	0%
Pothagam	0	0%
Tharpagam	5	12.5%
Santhigam	10	25%

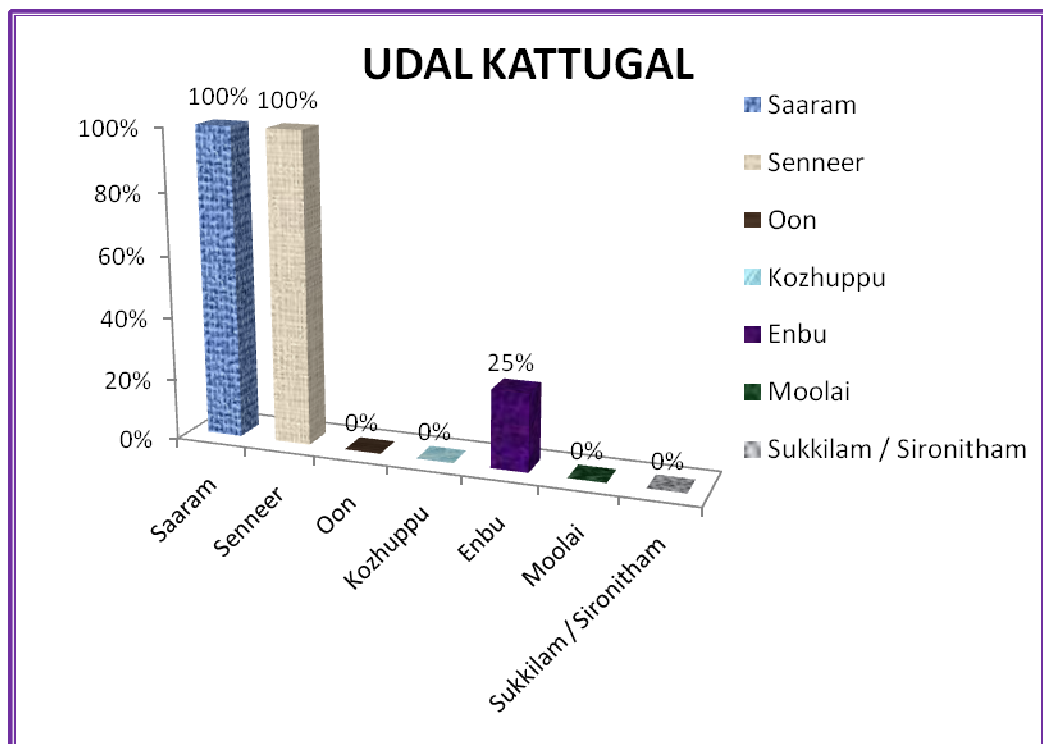


### Inference

According to Kabam Santhigam was deranged in 25%, Tharpagam was deranged in 12.5%, Avalambagam was deranged in 5% of cases.

## UDAL KATTUGAL

Udal Kattugal	No.of Cases Out of 40	Percentage %
Saaram	40	100%
Senneer	40	100%
Oon	0	0%
Kozhuppu	0	0%
Enbu	10	25%
Moolai	0	0%
Sukkilam / Sironitham	0	0%

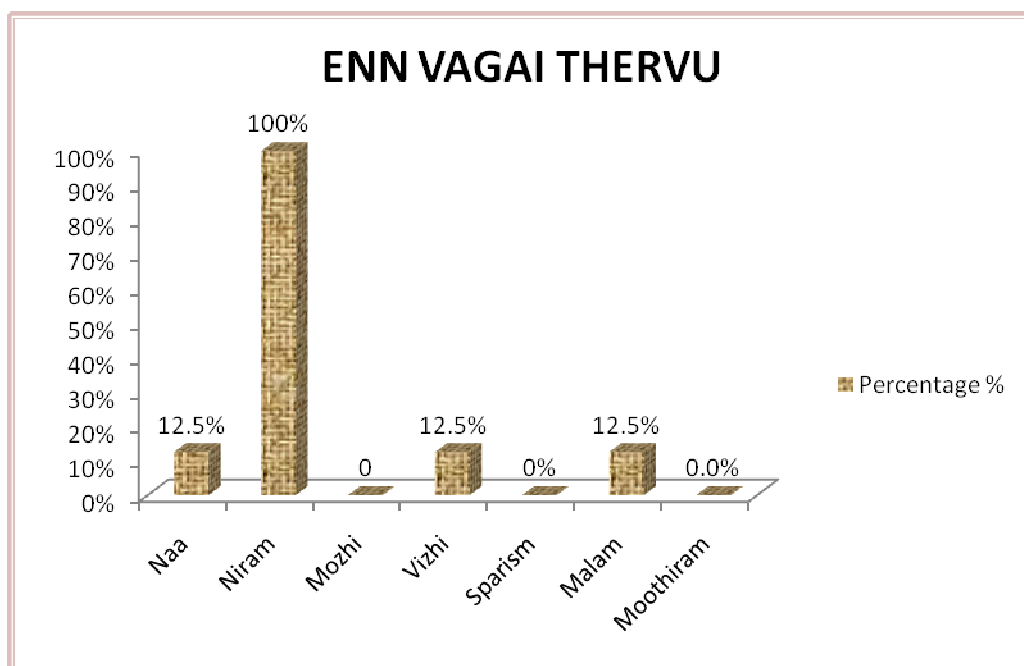


### Inference

Saaram, Senneer were affected in 100% of cases Enbu was affected in 25% .

## ENN VAGAI THERVU

Enn Vagai Thervu	No.of Cases Out of 40	Percentage %
Naa	5	12.5%
Niram	40	100%
Mozhi	0	0%
Vizhi	5	12.5%
Sparism	0	0%
Malam	5	12.5%
Moothiram	0	0%

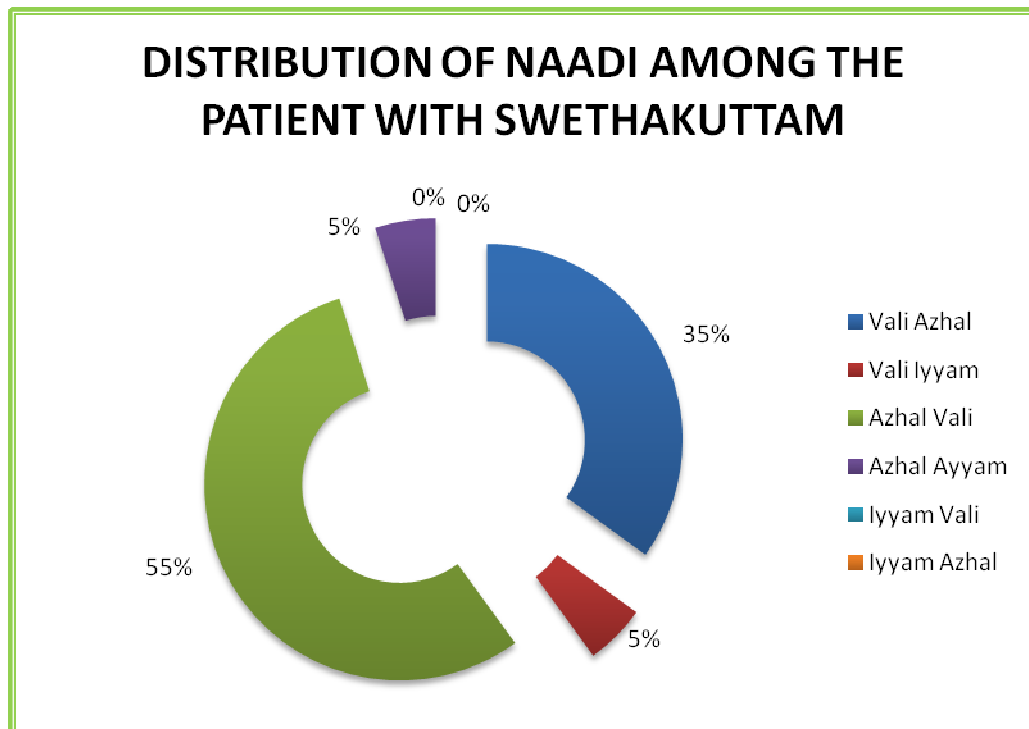


### Inference

Niram was affected in 100% of cases Naa, Vizhi, Malam were affected in 12.5% .

## DISTRIBUTION OF NAADI AMONG THE PATIENT WITH SWETHAKUTTAM

Naadi	No. of Cases Out of 40	Percentage %
Vali Azhal	14	35%
Vali Iyyam	2	5%
Azhal Vali	22	55%
Azhal Ayyam	2	5%
Iyyam Vali	0	0%
Iyyam Azhal	0	0%



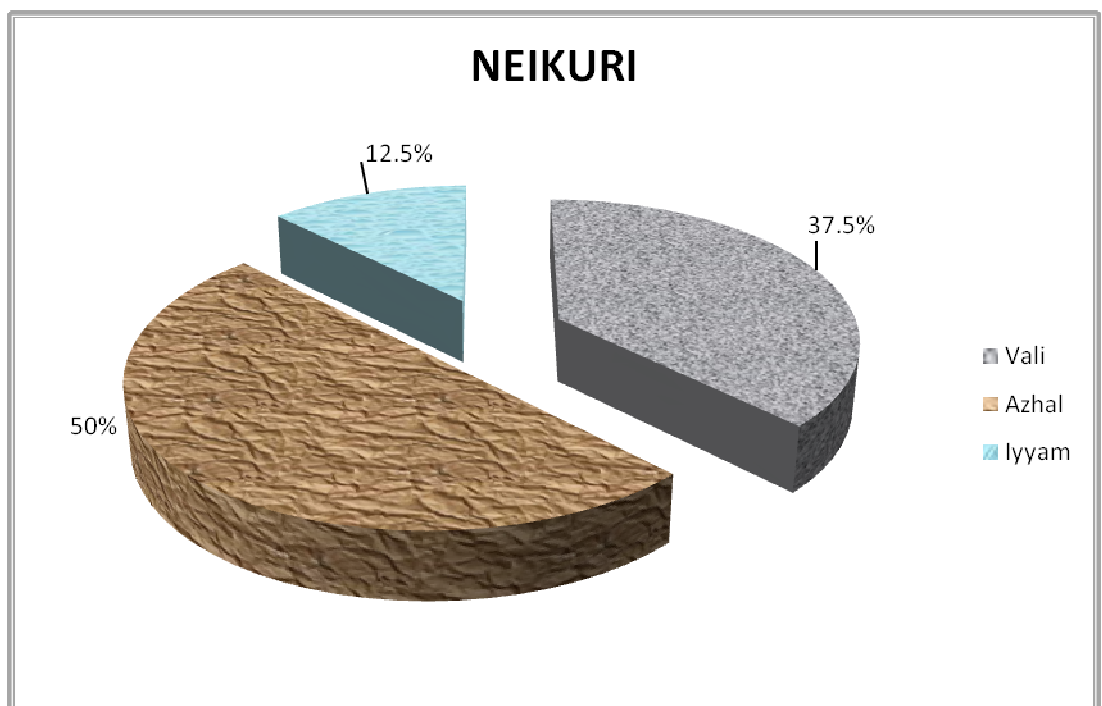
### Inference

Vali Azhal was observed in 35%, Azhal Vali was observed in 55%

Vali Ayyam was observed in 5%, Azhal Ayyam was observed in 5%

## NEIKURI REFERENCE

Neikuri	No.of Cases Out of 40	Percentage %
Vali (spreads like snake)	15	37.5%
Azhal (spreads like ring)	20	50%
Iyyam (stands like pearl)	5	12.5%



## Inference

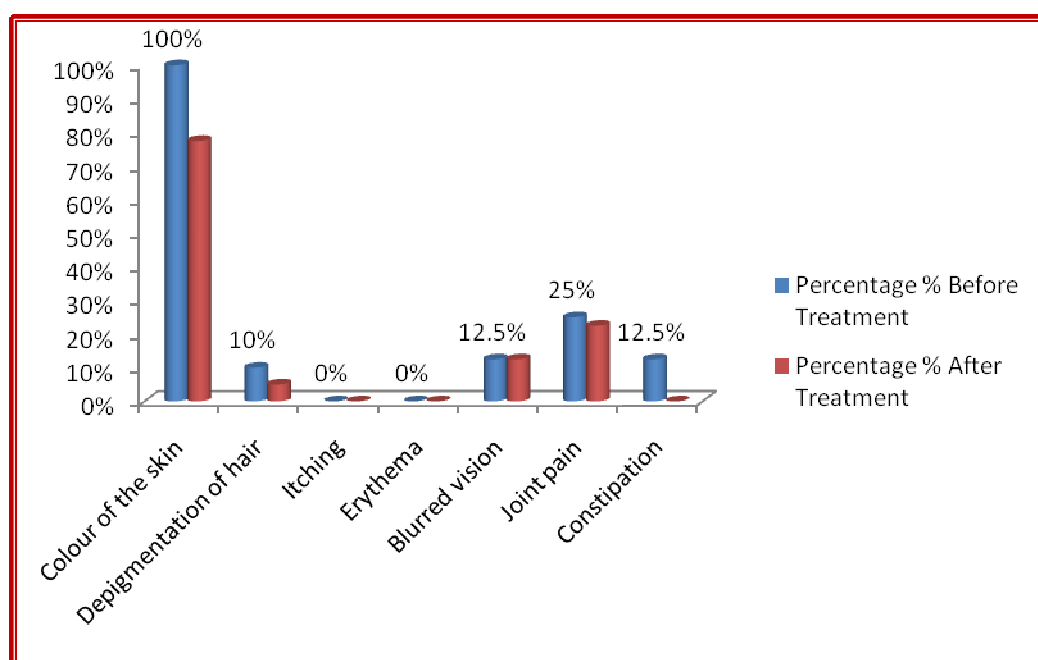
50% shows Azhal neikuri, 37.5% of cases show Vali neikuri and 12.5% Iyyam neikuri.



## CLINICAL FEATURES BEFORE & AFTER TREATMENT

Signs & Symptoms	No. of Cases Out of 40	Percentage % Before Treatment	No. of cases Improved from Signs & symptoms	Percentage % After Treatment
Colour of the skin	40	100%	31	77.5%
Depigmentation of hair	3	10%	2	5%
Itching	0	0%	0	0%
Erythema	0	0%	0	0%
<b>Other Features</b>				
Blurred vision	5	12.5%	5	12.5%
Joint pain	10	25%	9	22.5%
Constipation	5	12.5%	0	0%

## CLINICAL FEATURES BEFORE & AFTER TREATMENT



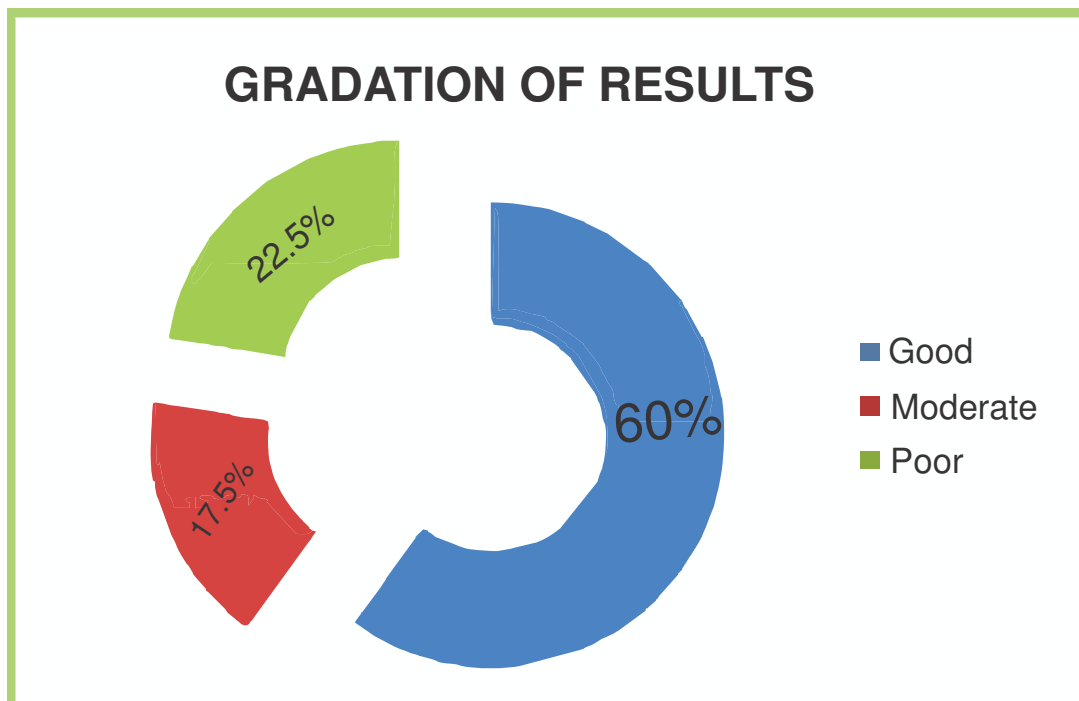
### Inference

Among 40 cases 31 cases are improved from this disease which has been noted as appearance of enormous repigmented spots in affected areas.

Among 3 cases 2 of them had improvement in colour change of hair from gray or white to normal colour or black among 10 cases 9 cases improved from joint pain constipation is relieved from noted cases.

## GRADATION OF RESULTS

Results	No.of Cases Out of 40	Percentage %
Good	24	60%
Moderate	9	22.5%
Poor	7	17.5%



### Inference

About 60% of cases had good Relief, 22.5% had moderate relief and 17.5% poor relief.

## HAEMOGLOBIN LEVEL BEFORE AND AFTER TREATMENT

Sl.No.	OP No.	Patients Name	Age	Haemoglobin Level gm %	
				Before Treatment	After Treatment
1	8700	Mrs. Meenammal	45	7	11.6
2	4646	Mrs. Gowri	24	10.8	12.6
3	4890	Mrs. Lalitha	52	9.6	11.7
4	4601	Mr. K.S.Kumar	20	9.0	12.2
5	1438	Master. Rohith	12	9.2	11.5
6	568	Ms. Lakshmi	19	8.0	11.6
7	1528	Mr. Ellappan	50	8.0	10.5
8	3091	Mr. Srinivasan	22	10.4	11.6
9	067	Mrs. Thaiyalnayaki	55	9.0	10.5
10	9057	Mrs. Madhuram	46	10.2	12.4
11	7099	Mr. Krishnamoorthi	50	10.5	12.6
12	490	Mr. Dhatchayani	50	10.6	13.2
13	9746	Mr. Jayakumar	49	10.0	13.1
14	344	Mr. Nainiyappan	50	9.6	12.4
15	487	Mr. Gopal	48	9.8	11.0
16	8387	Mr. Radhakrishnan	45	10.4	12.4
17	1205	Mrs. Kala	42	10.8	12.6
18	229	Master. Anil kumar	18	10.6	12.4
19	1230	Mrs. Sathiyabavani	27	9.8	11.2
20	4943	Mrs. Sowntharam	50	8.0	11.1

## DISCUSSION

Swetha Kuttam is an acquired idiopathic depigmentary condition and is characterized by completely depigmented macules and patches of varying sizes and shapes.

The clinical study of Swetha Kuttam was carried out post graduate department of Maruthuvan Govt. Siddha Medical College, attached to Arignar Anna Hospital of Indian Medicine, Arumbakkam, Chennai – 106.

40 patents of both sexes of various adult age groups satisfying the inclusion and exclusion criteria were selected. All necessary investigation was done for them and photographs of the lesion were taken. A day before starting the trial treatment, clearing of mukkutram by purgation will be carried out by Agasthiyar Kuzhambu – 100 mg, early morning with hot water. All the patients were treated with trial medicine. Hence with the help of trial medicines results and observation are noted for this study.

Let me make out these results on each category to arrive a better conclusion.

5 (12.5%) Patients were in the age group between 40-55 yrs.

12 (30%) Patients were in the age group between 10-30 yrs.

23 (57.5%) Patients were in the age group between 30-40 yrs.

### **Gender Distribution:**

40 patients of both the gender were selected for the dissertation study. Among the 26 (65%) were males, 14 (25%) were in females and the gender distribution was more or less equal.

**Socio Economic Status**

About 52.5% of the patients were middle income group (Rs.10001 to 20001) and 37.5% lower income (below Rs.10000) and 10% high income group above Rs.20000.

**Dietary Habits:**

28 (70%) Patients were taken mixed diet and only 12 (30%) taken vegetarian.

**Seasonal Variation:**

19 (47.5%) cases were admitted to trial in muthuvenil kaalam, 15 (37.5%) in Kaarkaalam, 4 (10%) Koothirkaalam, 2 (5%) Pinpani Kaalam, 8 (20%) Elavenil Kaalam.

**Etiology:**

About 33% unknown cases, 10% of the cases were of auto immune disease, and 7.5% hereditary cases.

**Thinai:**

29 (72.5%) Patients belonged to Neithal Thinai

5 (12.5%) Patients belonged to Marutham Thinai

4 (10%) Patients belonged to Kurunchi Thinai

2 (5%) Patients belonged to Mullai Thinai

In Sidha literature the Marutham is mentioned as disease free land among the five lands. Because of various environmental changes in the life style Swetha Kuttam occurs irrespective of the land, maximum patients came from in and around Chennai which belongs to Neithal Thinai.

## **Reference of Mukkutram**

### **1. Vaatham**

Viyanan was affected in 10 (25%), results in disability to move the joints

Pranan was affected in 2 (5%), results in dyspnoea.

Abaanan was affected in 5 (12.5%), results in constipation.

Koorman was affected in 5 (12.5%), results in visual impairment.

### **2. Pitham**

Praasakam was affected in 40 (100%) patients, results in hypopigmentation of skin.

Ranjagam was affected in 20 (50%) patients, results in decreased level of haemoglobin.

Alosagam was affected in 5 (12.5%) patients, results in visual impairment.

**Praasakam is responsible for the complexion of the skin. Due to defect in praasakam skin is changed in swethakuttam.**

### **3. Kabam**

Sandhigam was affected in 10 (25%) patients, results in disability to move the joints.

Avalambagam was affected in 2 (5%) patients, results in dyspnoea.

Tharpagam 5 (12.5%).

## **Udal Kattugal**

- Saaram was affected in all the 40 (100%) cases, results in tiredness
- Senneer was affected in 40 (100%) patients results in decreased level of haemoglobin.
- Enbu was affected in 10 patients (25%) results in restricted movements in both knee joints.

**Ennavagai Thervugal:**

- Niram was affected in all the (100%) cases because in Swetha Kuttam, the colour of skin changed into white.
- Naa was affected in 5 (12.5%) cases results in paleness of the tongue.
- Vizhi was affected 5 (12.5%) cases results in diminished vision.
- Malam was affected in 5 (12.5%) cases results in constipation.
- In Naadi, among 40 cases Vali Azal was noted in 35% of cases., Azalvali 22 (55%) and Azal Ayyam 2 (5%) .
- In Neikkuri, Pitha Neer was observed in 50% cases.

**Clinical Features before & after treatment**

Among 40 cases 33 cases are improved from this disease which has been noted as appearance of enormous repigmented spots in affected areas.

Rest of the 7 cases had slight repigmented spots in affected areas.

Among 3 cases of them had improvement in the colour change of hair from gray or white to normal colour or black.

**Trial Medicines:**

- I Puvarasam Pattai Kudineer Chooranam 25 gm- 60ml (Internal) twice a day
- II Nuna Thylam – 30 ml (External)

Patients were instructed to take the medicines regularly and apply the thylam twice a day and to expose the affected parts to sunlight. Diet restrictions were strictly imposed and followed.

**Mode of action of Trial Medicine:**

Swetha Kuttam is mainly due to vitiation of Azhal Kuttram, increased Azhal Kuttram is brought to normal by Enippu Suvai, Kaippu Suvai.

The trial drug consist of Thuvarpu Suvai and thus they decrease the azhal Kuttam by cool potency by nature.

So I conclude the Puvarasam Pattai Kudineer Chooranam cures the Swatha Kuttam and it comes under the **Ethirurai Maruthuvam**.

#### **Chemical Analysis:**

The chemical analysis of Puvarasam Pattai Kudineer Chooranam shows the presence of Iron, Zinc, Calcium, Potassium, Sugar, chloride.

#### **Pharamacological Report & Toxicological Evaluation:**

The drug also subjected to pharmacological and toxicological tests in rat models. The results revealed that the drug had very good haematinic effect. There were no signs of toxicity as could be judged by the absence of undersirable clinical manifestations.

#### **Bio Statistical Study:**

The bio-statistical report of the clinical trial shows significant result.



## SUMMARY

Swetha kuttam has been chosen for the dissertation work by the author .

Various literatures dealing with swetha kuttam have been collected from siddha and modern text books.

40 patients of both sexes, various adult age groups satisfying the inclusion and exclusion criteria were selected. All necessary investigation was done for them and photographs of the lesions were taken.

A day before starting the trial treatment, patients are given for neutralizing the mukkutram by purgation Agasthiyar kuzhambu 100mg early morning with hot water. All the patients were treated with trial medicines which are,

1. Puvarasampattai kudineer choornam
2. Nuna thylam

The result obtained from the studies are summarized below Male 26 (65%), were affected more than female 14 (35%). The cases were noted in the age group ranging from 10-55 yrs.

Middle classes 52.5% and lower 37.5% were more affected than upper classes 10%.

High incidence of swetha kuttam are found in neithal thinai 72.5% and muthuvenil kaalam 47.5%.

On examination of uyir thathukkal the following were deranged in more number of cases.

- |           |   |   |
|-----------|---|---|
| In vatham | : | Viyanan 25%, and koorman were affected 12.5%.               |
| In pitham | : | Prasagam 100%, ranjagam 100%, alosagam 12.5% were affected. |
| In kapham | : | Avalambagam 5%, santhigam 37.5% was affected.               |

In udal kattugal: Saaram 100%, seneer 100%, and enbu 25% were affected.

In envagai thervu: Naa 12.5%, niram 100%, vizhi 12.5%, malam 12.5% were affected.

Among the 40 patients responding to the trial medicines, 33 patients showed improvement which has been noted as appearance of chormous repigmented spots in affected areas. The rest of 7 patients had slight repigmentation.

Among 3 patients 2 of them had improvement in the colour change of the hair from gray or white to normal colour of the hair.

The chemical studies of the trial drugs possess, Iron, Zinc, Sugar Calcium, Potassium, Chloride.

The drug is also subjected to pharmacological and toxicological tests in rats as models. The results revealed that the drug had very effective results. There were no signs of toxicities that could be judged by the absence of undesirable clinical manifestations.

The bio-statistical report of the clinical trial shows significant result.

## CONCLUSION

- Swetha kuttam may occur due to various causes and it leads to mental stress and strain.hence,it is one of the cause of psychosomatic disorder.when the trial drug Puvarasampattai kudineer choornam (int),with Nuna thylum(ext) were administered to the swetha kuttam patients,it showed improvement in varying degrees in all the cases.
- In Swetha Kuttam Pitha Kutram is affected. The affected kuttram is neutralized by thuvarppu suvai. Thereby the trial medicine puvarasampattai kudinner choornam having the thuvarppu suvai acts on **Ethirurai** to cure the discease.
- In Chemical Analysis the trial medicine contain Iron, it is very essential to induce erythropoiesis.
- From the pharmacological study puvarasampattai kudineer choornam increase the haemoglobin level.
- The puvarasampattai kudineer choornam does not produce any toxicity in preclinically.so it is non-toxic and safe drug for swetha kuttam.
- No adverse effects were noticed during treatment period. The ingredient of puvarasampattai kudineer choornam of plant easily available and harmless to human being.

From this clinical studies,I conclude that the trial medicines which gives a 60% of improvement within minimum of 45 days to maximum of 90 days of treatment.further continuation of this medications for prolonged period of time may give complete cure in swetha kuttam.

## ANNEXURE –I

### CHEMICAL ANALYSIS OF TRIAL MEDICINES

**Preparation of Sodium Carbonate extract:** 2 gm of the sample is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No.	Experiment	Observation	Inference
1	Test for Acid Radicals		
a.	<b>Test for Sulphate</b> 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b.	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2.	<b>Test for Chloride:</b> 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	White precipitate is obtained	Present
3.	<b>Test for Phosphate</b> 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Absent of Yellow Precipitate.	Absent
4.	<b>Test for Carbonate:</b> 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absent
5.	<b>Test for Sulphide:</b> 1 gm of the substance is treated with 2ml of concentrated Hydrochloric acid	Absence of Rotten egg smelling	Absent

6.	<b>Test for Nitrate:</b> 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absent
7. a.	<b>Test for Fluoride and oxalate</b> 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absent
b.	5 drops of clear solution is added with 2ml of dilute sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of KMNO <sub>4</sub> solution discolourisation.	Absent
8.	<b>Test for Nitrite</b> 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent
9.	<b>Test for Borate</b> 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
<b>II.</b>	<b>TEST FOR BASIC RADICALS</b>		
10.	<b>Test for lead</b> 2 ml of the extract is added with 2 ml of Potassium iodide solution	Absence of Yellow precipitate	Absent
11a	<b>Test for Copper</b> One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absent of blue coloured flame.	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent

12.	<b>Test for Aluminium</b> To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
13a	<b>Test for Iron</b> To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Absence of Blood red colour	Absent
b.	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Absent of Blood red colour.	Absent
14.	<b>Test for Zinc</b> To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	White precipitate is obtained	Present
15.	<b>Test for Calcium</b> 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	White precipitate is obtained	Present
16.	<b>Test for Magnesium</b> 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
17.	<b>Test for Ammonium</b> 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absent of Reddish brown precipitate	Absent
18.	<b>Test for Potassium</b> A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Yellow precipitate is obtained	Present
19.	<b>Test for Sodium</b> 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent

20.	<b>Test for Mercury</b> 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent
21.	<b>Test for Arsenic</b> 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absence of Yellow precipitate.	Absent
22.	<b>Test for Starch</b> 2ml of extract is treated with weak iodine solution	Absent of Bluecolour.	Absent
23.	<b>Test of reducing Sugar</b> 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Green colour is obtained.	Present
24.	<b>Test of the alkalioids</b> 2ml of the extract is treated with 2ml of potassium Iodide solution	Absent of Red colour	Absent

## RESULTS:

The given sample contains.  
(Puvarasampattai Kudineer Choranam)  
Chloride, Zinc, Sugar, Iron.

# **TOXICITY STUDY ON POOVARASAM PATTAI KUDINEER CHOORANAM**

## ***Animals***

Mice of either sex weighing 25-30g and rats weighing 155-174g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28<sup>0</sup>C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

## **ACUTE TOXICITY STUDY-OECD 425 GUIDELINES**

Acute oral toxicity test for the Poovarasam Pattai Kudineer Chooranam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice.

The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the



next dose was delayed until one is confident of survival of the previously dosed animal.

***Observation of toxicity signs:*** General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

### **SUB-ACUTE TOXICITY**

The 28 days repeated dose oral toxicity study was carried out on twenty four rats and were divided into 4 groups of 6 in each. The rats received daily control (2% CMC 2ml/kg) and 100, 200, 400mg/kg/day Poovarasam Pattai Kudineer Chooranam by oral gavage, once daily for 28 consecutive days. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity.

The body weight changes, food and water consumption was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at 4°C for 10 min to separate the serum and used for the biochemical assays.

### ***Hematological and blood biochemical analyses:***

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis glucose, creatinine, total protein, albumin, total

and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

#### ***Necropsy:***

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

#### **Statistical analysis**

Values were represented as mean  $\pm$  SEM. Data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparison test using GraphPad Instat-V3 software. P values  $< 0.05$  were considered significant.

### **RESULTS**

All the animals from control and all the treated dose groups up to 400 mg/kg survived throughout the dosing period of 28 days. No signs of major or significant intoxication were observed in animals from lower to higher dose groups during the dosing period of 28 days. Animals from all the treated dose groups exhibited comparable body weight gain with that of controls throughout the dosing period of 28 days. Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days. Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality.

The results of haematological investigations revealed no significant changes in the values of different parameters investigated when compared with those of respective controls except RBC and Hb ( $P < 0.01$ ) count. A slight increase in total RBC count values were obtained for animals in the dose group of 400 mg/kg only. Results of Biochemical investigations revealed no significant changes in the values of different parameters studied when compared with those of respective controls; however, the values obtained were within normal biological and laboratory limits. Functional observation tests conducted at termination revealed no abnormalities.

Urine analysis, conducted at the end of the dosing period in week 4 revealed no abnormality attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Gross pathological examination did not reveal any abnormality. Histopathological examination did not reveal any abnormality.

## **CONCLUSION**

No mortality, no treatment related clinical signs were observed throughout the study period. In respect of initial body weight, there was increase of body weight and there were no statistical changes in body weights in any groups as compared to control animals. So, these changes in rats had no biological significance. No other treatment-related changes in clinical chemistry parameters. Gross necropsy examination revealed no treatment-related lesions. Hence, it can be concluded that the drug Poovarasam Pattai Kudineer Chooranam is well tolerable and practically non toxic in animal models upto 400mg/kg dose level and can be used clinically for various ameliorations.

## REFERENCES

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**Table 5. Body wt (g) of rats exposed to Poovaram Pattai Kudineer Chooranam for 28days.**

<b>Dose (mg/kg/day)</b>	<b><i>Days</i></b>				
	<b>1</b>	<b>7</b>	<b>14</b>	<b>21</b>	<b>28</b>
<b>Control</b>	174.42±5.26	178.49±4.18	181.22±5.22	186.42±4.70	188.12±5.44
<b>100</b>	158.34±4.20	163.55±5.24	165.18±5.80	168.54±5.00	176.52±4.10*
<b>200</b>	167.10±4.17	169.00±4.40	172.04±5.33	174.10±4.15	176.62±5.65
<b>400</b>	155.19±5.23	158.00±5.65	160.12±4.38	161.58±5.47	165.78±4.30

Values are mean ± S.E.M. (Dunnet 't' test). \*P<0.05. Vs. Control group N=6.

**Table 6. Food (g/day) intake of rats exposed to Poovaram Pattai Kudineer Chooranam for 28days.**

Dose (mg/kg/day)	Days (gms/rats)				
	1	7	14	21	28
<b>Control</b>	47.02±2.52	45.21±2.48	45.12±2.65	48.14±2.54	45.11±3.00
<b>100</b>	44.28±2.60	44.40±2.17	45.42±2.45	46.10±2.26	46.19±3.00
<b>200</b>	46.30±2.38	44.56±2.10	44.64±2.64	45.45±2.14	45.01±2.88
<b>400</b>	45.15±2.26	45.22±2.85	48.10±2.20	47.23±2.55	49.20±2.18

Values are mean ± S.E.M. (Dunnet 't' test). <sup>ns</sup>P>0.05. Vs. Control group N=6.

**Table 7. Water (ml/day) intake of rats exposed to Poovaram Pattai Kudineer Chooranam for 28days.**

Dose (mg/kg/day)	Days(ml/rat)				
	1	7	14	21	28
<b>Control</b>	51.26±2.55	52.66±3.30	54.15±3.16	52.52±3.10	51.00±3.50
<b>100</b>	50.14±2.14	50.10±3.85	55.20±4.49	48.12±3.18	48.55±2.95
<b>200</b>	48.07±2.23	45.21±3.58	48.58±3.34	46.18±2.46	49.44±3.28
<b>400</b>	50.00±3.45	52.76±3.50	50.30±3.15	47.25±3.12	50.58±3.52

Values are mean ± S.E.M. (Dunnet 't' test). <sup>ns</sup>P>0.05. Vs. Control group N=6.

**Table 8. Hematological parameters after 28days treatment with Poovaram Pattai Kudineer Chooranam in rats.**

<b>Parameter</b>	<b>Control</b>	<b>100 mg/kg</b>	<b>200 mg/kg</b>	<b>400 mg/kg</b>
<b>Red blood cell (mm<sup>3</sup>)</b>	15.00±0.50	14.90±0.58	15.15±0.47	16.28±0.52
<b>HB (%)</b>	14.18±0.36	15.21±0.30	16.57±0.42**	17.22±0.40**
<b>Leukocyte (x10<sup>3</sup>/Cu.mm)</b>	8.20±1.7	8.24±0.82	8.12±1.10	8.32±1.31
<b>Platelets(K/μl)</b>	442±20.18	456±30.42	447±30.20	472±33.46
<b>MCV (gl)</b>	52.40±4.81	52.28±4.38	53.08±3.32	52.18±4.00
<b>N</b>	45.45±1.38	45.20±1.12	41.84±0.82	45.10±3.14
<b>L</b>	55.12±2.42	54.84±3.41	53.28±3.52	55.12±3.48
<b>M</b>	1.40±0.30	1.40±0.31	1.40±0.29	1.38±0.22
<b>E</b>	1.00±0.00	1.00±0.12	1.00±0.11	1.00±0.10
<b>B</b>	0±0.00	0±0.00	0±0.00	0±0.00
<b>ESR(mm)</b>	1±00	1±00	1±00	1±00
<b>PCV</b>	45.55±2.60	45.08±2.54	45.10±3.00	45.20±3.02

Values are mean ± S.E.M. (Dunnet 't' test). \*\*P<0.01. Vs. Control group N=6.

**Table 9. Effect of treatment with Poovaram Pattai Kudineer Chooranam biochemical parameters.**

<b>Dose (mg/kg)</b>	<b>Control</b>	<b>100 mg/kg</b>	<b>200 mg/kg</b>	<b>400 mg/kg</b>
<b>Total Bilirubin (mg/dL)</b>	0.27±0.05	0.26±0.06	0.25±0.05	0.25±0.04
<b>Bilirubin direct (mg/dL)</b>	0.21±0.07	0.21±0.09	0.20±0.05	0.21±0.06
<b>ALP (U/L)</b>	102.22±7.12	105.14±8.00	105.10±7.24	104.48±9.20
<b>SGOT (U/L)</b>	114.24±5.18	112.32±4.50	110.51±5.22	112.00±4.26
<b>SGPT(U/L)</b>	35.14±2.00	34.72±2.04	35.82±2.21	34.23±2.18
<b>Total Protein(g/dl)</b>	7.00±1.32	7.14±1.21	7.54±0.22	8.00±0.23
<b>Albumin(g/dl)</b>	3.15±0.20	3.18±0.21	3.40±0.20	3.42±0.22
<b>Globulin(g/dl)</b>	5.00±0.11	5.10±0.12	4.88±0.10	4.84±0.12

Values are mean of 6 animals ± S.E.M. <sup>ns</sup>P>0.05. Vs. Control group N=6.



**Table-10 RFT**

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Urea(mg/dL)	5.36±1.85	4.80±2.10	5.0±2.00	5.11±1.45
Creatinine (mg/dL)	0.71±0.06	0.70±0.05	0.71±0.06	0.71±0.05
Uric acid (mg/dL)	3.51±0.17	4.00±0.12*	4.10±0.12*	4.00±0.11*
Na m.mol	116.85±5.16	115.2±5.45	117.42±5.00	117.14±5.12
K m.mol	5.22±2.81	5.52±1.10	5.10±1.11	6.12±2.02
Cl m.mol	100.08±4.10	101.00±5.14	99.16±4.00	100.21±5.18

Values are mean ± S.E.M. \*P<0.05. Vs. Control group N=6.

**Table-11. Lipid Profile**

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Total cholestrol(mg/dL)	78.15±2.52	74.18±2.44	75.12±3.00	76.00±2.10
HDL(mg/dL)	125.00±2.54	121.42±2.70	124.14±3.52	125.42±2.28
LDL(mg/dL)	42.55±2.32	42.58±3.12	41.15±2.80	42.00±3.00
VLDL(mg/dl)	25.30±2.28	25.48±2.15	25.04±2.44	25.00±2.22
Triglycerides (mg/dl)	28.42±3.00	27.51±2.40	26.88±3.12	27.15±2.74
Blood glucose(mg/dl)	95.15±4.25	95.12±4.05	94.14±5.04	94.54±2.41

Values are mean ± S.E.M. <sup>ns</sup>P>0.05. Vs. Control group N=6.

Table-12 Urine Analysis

Parameters	Control	100 mg/kg	200 mg/kg	400 mg/kg
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>7.5	>7.5
Protein	Nil	1+	1+	2+
Glucose	Nil	Nil	Nil	Trace
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	-ve	-ve	-ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Normal	Normal	Normal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

**Table 13. Effect of oral administration of Poovaram Pattai Kudineer Chooranam on organ weight**

<b>Dose (mg/kg)</b>	<b>Control</b>	<b>100 mg/kg</b>	<b>200 mg/kg</b>	<b>400 mg/kg</b>
<b>Liver (g)</b>	3.12±0.10	3.10±0.15	3.15±0.12	3.15±0.15
<b>Heart (g)</b>	0.34±0.04	0.33±0.05	0.35±0.04	0.34±0.04
<b>Lung (g)</b>	0.45±0.16	0.44±0.16	0.43±0.15	0.44±0.15
<b>Spleen (g)</b>	0.45±0.05	0.45±0.04	0.46±0.04	0.45±0.05
<b>Ovary (g)</b>	1.20±0.16	1.22±0.15	1.22±0.15	1.24±0.14
<b>Testes (g)</b>	2.38±0.11	2.41±0.20	2.40±0.22	2.40±0.20
<b>Brain (g)</b>	2.00±0.12	2.04±0.10	2.06±0.12	2.32±0.14
<b>Kidney (g)</b>	0.80±0.04	0.81±0.04	0.80±0.04	0.82±0.05
<b>Stomach (g)</b>	1.21±0.12	1.24±0.10	1.22±0.12	1.21±0.10

Values are mean of 6 animals ± S.E.M. (Dunnet 't' test). <sup>ns</sup>P>0.05 Vs. Control group N=6.

## **HAEMATINIC ACTIVITY OF POOVARASAM PATTAI KUDINEER CHOORANAM IN PHENYLHYDRAZINE INDUCED ANEMIC RATS**

### **INTRODUCTION**

Anaemia is very common and the incidence is likely to increase in future, there is need to prevent it or seek for more cost-effective and better treatment strategies. Iron plays a significant role in the haematopoiesis. However, the therapeutic potential of the herbs cannot be established on the basis of available iron content alone as other factors play a role in the absorption of iron in the body. Excess doses of iron by ingestion are a pain in stomach due to lining of ulceration. This is accompanied by nausea and vomiting. The iron may pass deeper into the body and damages internal organs particularly the brain, liver and metabolic acidosis develops. The pragmatic use of different formulation of native iron in the treatment of anemia is time of immemorial. Man throughout the ages has depended on his immediate environment for food and medication. Most especially, man has consistently resorted to plants for solution to the myriad of health problems challenging him.

The *Thespesia populnea* belongs to Malvaceae family is a fairly large, quick-growing evergreen tree distributed mainly along the coastal regions throughout India. The parts of this plant are used externally in scabies, psoriasis and other skin diseases. The plant is astringent, acrid, depurative, haemostatic, antidiarrheal and antibacterial. (+)-Gossypol was reported to be present in the bark of the plant and it was found to be optically active.

Traditional herbal practitioners have made several claims on numerous herbal preparations with specific claim on the efficacy of parts of *Thespesia papulnea* in the treatment of several disease conditions. According to the literature, the improvement in the haemoglobin level in these diseased conditions will enhance the beneficial treatment outcome. Hence in the present investigation, the

traditionally used siddha medicament Poovarasam Pattai Kudineer Chooranam was evaluated for its antianemic property in terms of haematinic action in scientific manner using phenylhydrazine induced anemic animal model.

## **MATERIALS AND METHODS**

### **Drug material**

Poovarasam Pattai Kudineer Chooranam was collected from siddha commercial lab and 2gms of this chooranam was suspended in 10ml of 2% CMC solution to achieve 200mg/ml stock solution and used in this study. The resulting suspension was then filtered. The filtrate was stored in a refrigerator until use.

### **Animals**

Male albino rats (150-180g) and Mice of either sex weighing 25-30g were used for the study. The rats were housed in wire-mesh cages with a 12 h light/dark cycle. They had continuous access to food and water during the entire period of experimentation.

### **Acute toxicity study**

Single dose acute oral toxicity test for the Poovarasam Pattai Kudineer Chooranam was carried out as per OECD Guidelines 425. Animals were observed individually after dosing at least once during the first 30 minutes, periodically during the first 24 hours, with special attention given during the first 4 hours, and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead.

All observations are systematically recorded and Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behaviour pattern. Attention was directed to observations of tremors, convulsions, salivation, diarrhoea, lethargy, sleep and coma. The principles and criteria summarised in the

Humane Endpoints Guidance Document taken into consideration. Animals found in a moribund condition and animals showing severe pain or enduring signs of severe distress was humanely killed.

### **Evaluation of Haematinic Activity**

Six rats were kept as normal control group (Group 1), while 24 rats were made anaemic by oral intubations of phenylhydrazine (10 mg/kg body weight) daily for 8 days. Rats that developed anaemia with haemoglobin concentration <14 g/dl were recruited for the study. Anaemic rats were randomly divided into 5 groups (2 to 6) and treated as follows: Group 1: received distilled water (1 ml) daily (normal control), Group 2: received 2% CMC (1 ml) daily (anaemic control), Group 3: received oral single dose of the Poovarasam Pattai Kudineer Chooranam 100mg/kg body weight/day Group 4: received oral single dose of the Poovarasam Pattai Kudineer Chooranam 200mg/kg, Group 5: received oral single dose of the Poovarasam Pattai Kudineer Chooranam 400mg/kg Group 6: received oral single dose of the heamatinic syrup 2ml/kg body weight/day. The treatment was continued for 2 weeks.

### **Haematological investigation**

Blood was collected from the retro orbital vein of experimental animals after an overnight fast (T=0) and after 1 and 2 weeks of treatment with Poovarasam Pattai Kudineer Chooranam, was used for the determination of red blood cell count (RBC), haemoglobin (Hb) concentration and packed cell volume (PCV). The mean cell volume (MCV), mean cell haemoglobin (MCH) and the mean cell haemoglobin concentration (MCHC) were calculated.

### **Statistical analysis**

Experimental data was analysed using analysis of variance (ANOVA) and Dunnet's 't' test to determine significant differences between means. The statistical analysis system (INSTAT-V3) package was used for this analysis.

## RESULTS AND DISCUSSION

Anaemia is one of the most widespread disorders of blood which affect the populations of all ages throughout the world. It is a public health problem that affects populations in both rich and poor countries. However, the incidence of this disorder is higher in the developing countries than in the developed countries due to poverty, malnutrition and lack of hygiene. The situation is aggravated by factors such as nutritional deficiencies and high prevalence of parasitic gastrointestinal infections which cause heavy loss of blood. It has been confirmed previously that intraperitoneal administration of phenylhydrazine decreased haemoglobin concentration, red blood cells number and haematocrit values.

The phytochemical screening of Poovarasam Pattai Kudineer Chooranam revealed presence of alkaloids, flavonoids, saponins and terpenoids. The acute toxicity testing revealed no significant toxic signs or death up to doses of 2000 mg/kg. In the untreated control rats phenylhydrazine induced significant ( $p < 0.05$ ) decrease in Hb concentration, RBC, WBC level, indicating anaemia. The administration of the Poovarasam Pattai Kudineer Chooranam produced a significant ( $p < 0.05$ ) increase in the haematological parameters. The phenylhydrazine induced anaemia was significantly ( $p < 0.05$ ) reversed after 14 days treatment with the Poovarasam Pattai Kudineer Chooranam at the dose level of 400mg/kg towards almost normal. In the anemic control, the Hb was  $12.21 \pm 1.34$ g/dl at day seven and this was improved to  $13.00 \pm 0.42$ ,  $15.36 \pm 1.15$  and  $15.87 \pm 1.26$ g/dl at the dose levels 100, 200 and 400mg/kg respectively. Similarly, After 14 days of treatment with Poovarasam Pattai Kudineer Chooranam 400mg/kg, the Hb level was increased from  $10.12 \pm 1.00$ g/dl to  $13.11 \pm 1.05$ ,  $14.52 \pm 1.92$  and  $16.33 \pm 1.53$ g/dl at the doses described earlier. Same kind of beneficial and significant ( $p < 0.05$ ) changes were recorded in the other haematological parameters and at the higher dose of the Poovarasam Pattai Kudineer Chooranam. The effect of commercially availed heamatinic syrup was comparable to those of the test drug. There was no change in other blood parameters like MCV, MCHC and MCH. On other hand, red blood cell count ( $p < 0.01$ ) in animals treated with 400mg/kg of Poovarasam Pattai

Kudineer Chooranam exhibit a statistically significant elevation when compared with control group. The main importance of this study is to correlate the positive or beneficial effects along with the cofactors like flavonoids and alkaloids in the treatment of various traditional claims of this drug. Factors like alkaloids, flavonoids, saponins, tannins, calcium, zinc, vitamins C and K are involving in the utilization of iron content by our body tissues. Especially vitamin C contributes to the bioavailability of iron in the body. Since, anemia causes important physiologic effects on the cardiovascular system, hormonal and metabolic effects can result in direct myocardial toxicity, myocardial hypertrophy, and salt and water retention, which could be harmful in patients with heart failure.

Scientific studies failed to give any valid evidence of an association between circulating hemoglobin level and the severity of symptoms. Number of other conditions, such as malaria and haemoglobinopathies are also responsible because the RBC plays a primary role of transporting substances including nutrients, respiratory gases and other waste materials throughout the body. The WBC defends the body against pathogens and other foreign bodies. The platelets play the role of preventing blood loss. Therefore, severe alteration in the concentration of any of these haemopoietic components may be detrimental.

At the nutritional level, the most current and primary cause of anaemia is the iron deficiency. A deficiency state can only be recognized, however, if there is unequivocal evidence of impairment to health. Values of biochemical or hematological variates that are lower than average, or lower than an arbitrarily chosen lower limit of normal, cannot be considered valid evidence of pathological deficiency, nor can an elevation of levels induced by increased intakes of a nutrient be accepted as evidence of an improvement in health. Approximately 30% of the world population are affected by an iron deficiency, making iron by far the most widespread nutrient deficiency worldwide.



About half of the iron deficient people suffer from the more severe form of iron deficiency anaemia. Anaemia has economic implications for the country because it can have profound effects on work performance and productivity. The people at the risk are the old persons, the young women in age to procreate and children. Iron deficiency anaemia can cause adverse pregnancy outcome, decrease immune function and has been recognized as an important cause of cognitive deficit in infants and young children. Despite the clinical belief that anemia causes symptoms, there seems to be little valid evidence of an increase in symptoms with low levels of circulating hemoglobin, or of a beneficial effect of iron therapy on wellbeing.

## **CONCLUSION**

From the toxicity study, no treatment related toxic signs and symptoms was observed and the Poovarasam Pattai Kudineer Chooranam, at a dose of 400 mg/kg (p.o.), significantly increased the haemoglobin, and RBC count in anaemic rats indicating the haematinic effect. The presence of alkaloids, saponins, tannins was confirmed by the preliminary phytochemical study. These phytochemicals could be responsible for their anti-anaemic effects.

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**Table 1: Dose finding experiment and its behavioral Signs of Toxicity**

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	1000	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	2000	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3	5000	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	+	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis

14. Analgesia 15. Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

**Table 2: Effect of Phenylhydrazine (10mg/kg, p.o. daily for 7 days) alone on Hematological parameters.**

<b>Parameters</b>	<b>Group 1 (Control)</b>	<b>Group 2 (anemic)</b>	<b>Group 3 (anemic)</b>	<b>Group 4 (anemic)</b>	<b>Group 5 (anemic)</b>	<b>Group 6 (anemic)</b>
<b>Hb (g/dl)</b>	18.00 ± 0.48	13.21±0.32**	13.56±0.24**	13.41±0.36**	14.12±0.31**	13.55 ± 0.29**
<b>PCV (%)</b>	56.42 ±1.66	41.92 ± 2.74**	40.28 ± 2.26**	41.22 ± 2.44**	40.51 ± 2.15**	40.34 ± 2.48**
<b>RBC (x10<sup>6</sup>/ml)</b>	6.30 ± 0.21	4.28 ± 0.24**	4.28 ± 0.21**	4.54 ± 0.25**	4.47 ± 0.20**	4.20 ± 0.32**
<b>MCV (fl)</b>	74.11 ± 2.72	82.45±4.02	86.18±5.35	85.55±4.24	86.11±5.65	81.99± 4.26
<b>MCH (pg)</b>	22.65 ± 1.56	29.18±1.85	31.45±2.52*	30.15±2.28	30.12±1.88	30.00 ± 2.23
<b>MCHC (g/dl)</b>	35.10 ± 0.52	33.34±0.72	33.27±0.80	34.11±0.98	34.15±1.06	30.75 ± 1.00*

Values are mean of 6 animals ± S.E.M. (Dunnet's test). \*P<0.05; \*\*P<0.01 Vs Control; \*P<0.05; \*\*P<0.01 Normal Vs Control..

**Table 3: Hematological parameter of rats after Seven days treatment with Poovarasam Pattai Kudineer.**

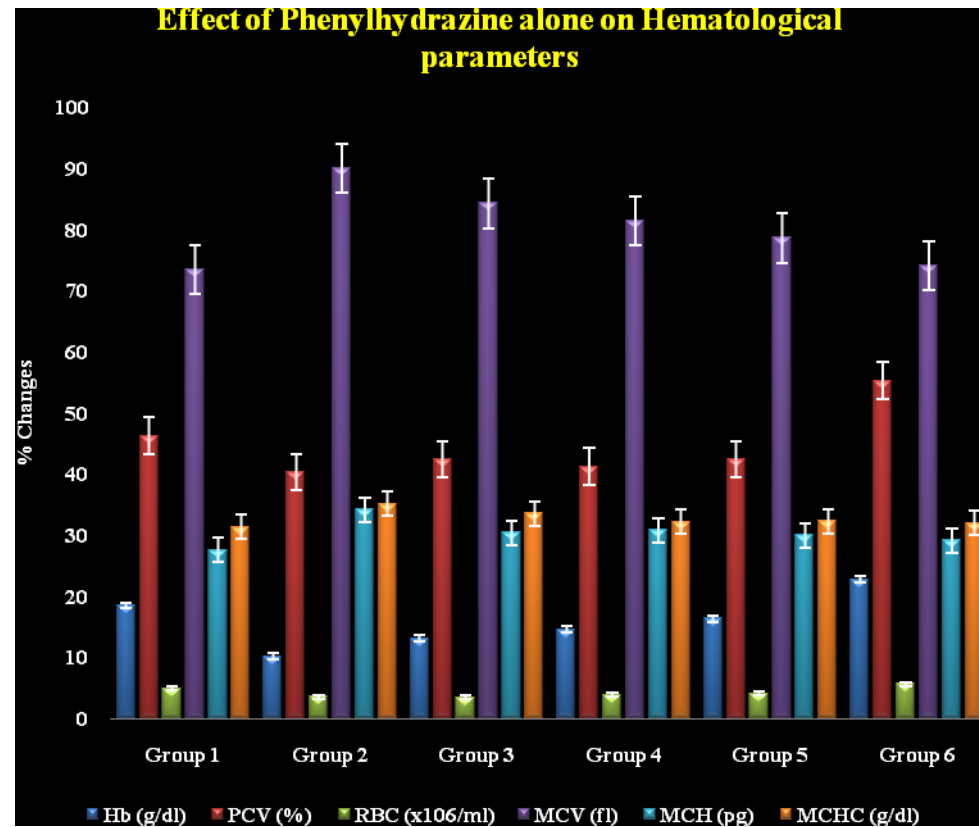
<b>Parameters</b>	<b>Group 1 (control)</b>	<b>Group 2 (anemic control)</b>	<b>Group 3 (5ml/kg)</b>	<b>Group 4 (10ml/kg)</b>	<b>Group 5 (20ml/kg)</b>	<b>Group 6 (Heamatinic syrup)</b>
<b>Hb (g/dl)</b>	18.56 ±1.33	12.21±1.34**	13.00±0.42*	15.36±1.15	15.87 ± 1.26	20.52 ±1.88
<b>PCV (%)</b>	52.18 ±1.22	43.85±2.26	46.74±2.41	47.00±2.48	49.39±2.98	54.65 ±2.61
<b>RBC (x10<sup>6</sup>/ml)</b>	7.62±0.28	5.55±0.20**	6.29±0.14**	6.54±0.17**	7.28±0.15	8.55±0.19**
<b>MCV (fl)</b>	77.24±2.22	78.10±2.47	80.32±2.83	80.12±2.14	80.00±3.00	76.52±2.68
<b>MCH (pg)</b>	25.88±1.74	28.1±1.52	23.20±1.41	24.16±1.56	24.22±1.19	26.24±1.50
<b>MCHC (g/dl)</b>	34.26±1.33	32.05±1.24	27.64±1.65*	26.00±1.21*	29.54±1.75	30.42±2.00

Values are mean of 6 animals ± S.E.M. (Dunnet's test). \*P<0.05; \*\*P<0.01 Vs Control; \*P<0.05; \*\*P<0.01 Normal Vs Control..

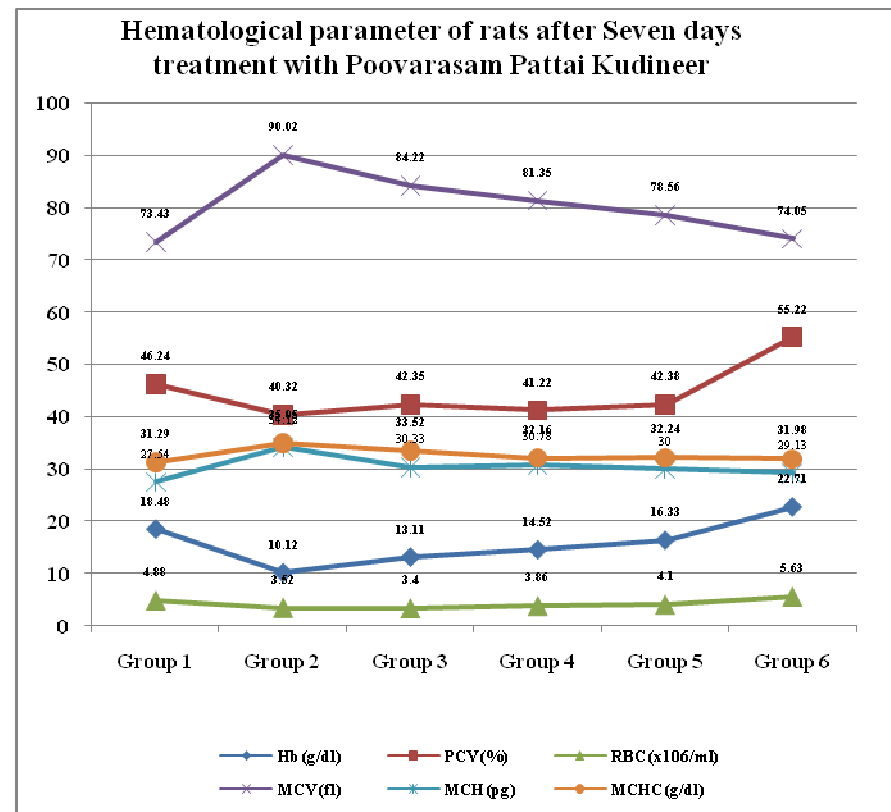
**Table 5: Hematological parameters of rats after 14 days treatment with Poovarasam Pattai Kudineer.**

<b>Parameters</b>	<b>Group 1 (control)</b>	<b>Group 2 (anemic control)</b>	<b>Group 3 (5ml/kg)</b>	<b>Group 4 (10ml/kg)</b>	<b>Group 5 (20ml/kg)</b>	<b>Group 6 (Heamatinic syrup)</b>
<b>Hb (g/dl)</b>	18.48±1.85	10.12±1.00**	13.11±1.05	14.52±1.92	16.33±1.53	22.71±2.04
<b>PCV (%)</b>	46.24±1.27	40.32±1.28*	42.35±1.62	41.22±1.38	42.38±1.12	55.22±1.32**
<b>RBC (x10<sup>6</sup>/ml)</b>	4.88±0.26	3.52±0.30*	3.40±0.22**	3.86±0.31	4.10±0.36	5.63±0.38
<b>MCV (fl)</b>	73.43±2.52	90.02±2.45**	84.22±2.12**	81.35±1.62	78.56±2.19	74.05±2.37
<b>MCH (pg)</b>	27.54±1.61	34.12±1.69	30.33±1.56	30.78±2.04	30.00±2.11	29.13±2.82
<b>MCHC (g/dl)</b>	31.29±2.45	35.05±1.32	33.52±1.08	32.16±1.12	32.24±2.00	31.98±2.28

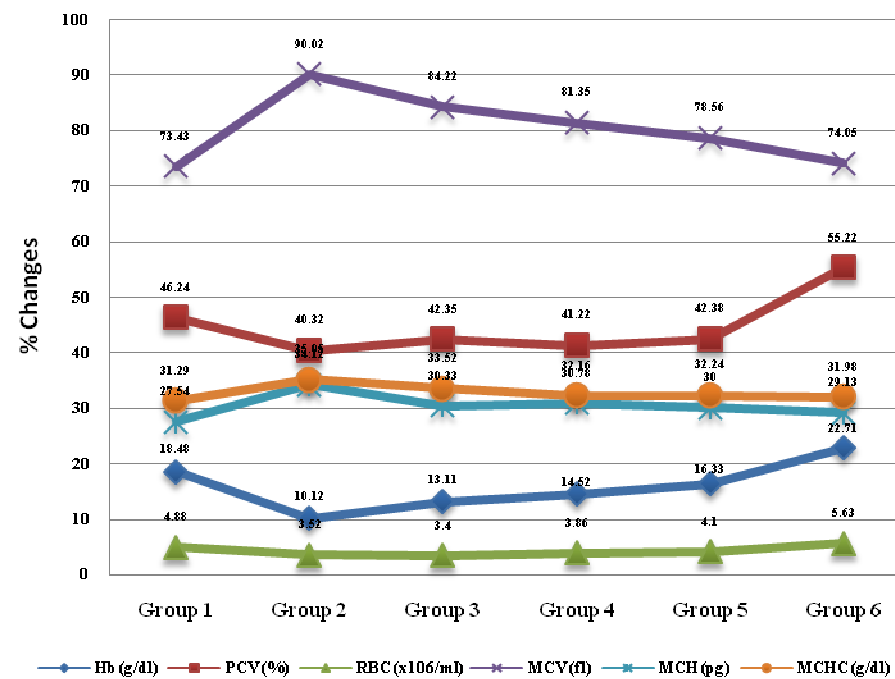
Values are mean of 6 animals ± S.E.M. (Dunnet's test). \*P<0.05; \*\*P<0.01 Vs Control; \*P<0.05; \*\*P<0.01 Normal Vs Control.







### Hematological parameters of rats after 14 days treatment with Poovarasam Pattai Kudineer



## BIOSTATISTICAL ANALYSIS

**Effect of Puvarasampattai Kudineer Chooranam on Hb level (gm%) in human subjects**

S.No	Haemoglobin Level gm%	
	Before Treatment	After Treatment
1	7	11.6
2	10.8	12.6
3	9.6	11.7
4	9.0	12.2
5	9.2	11.5
6	8.0	11.6
7	8.0	10.5
8	10.4	11.6
9	9.0	10.5
10	10.2	12.4
11	10.5	12.6
12	10.6	13.2
13	10.0	13.1
14	9.6	12.4
15	9.8	11.0
16	10.4	12.4
17	10.8	12.6
18	10.6	12.4
19	9.8	11.2
20	8.0	11.1

**Software:** spss17 version

**Variables:** Hb levels (gm %) – before treatment, after treatment

**Number of cases:** 20

**Test:** Paired t test

**Confidence Interval:** 95%

**Correlation coefficient (r):** 0.624

**Before and after treatment mean difference:**  $2.34 \pm 0.86$  (gm %)

**P Value (2 tailed):**  $p < 0.01$

### **Inference:**

The p value is significant ( $p < 0.01$ ). So the treatment was significantly improving the Hb level (mg%).

### **Treatment for Swetha kuttam**

The most popular statistical tool, namely, Fisher's Exact Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

### **Hypothesis**

There is no reducing symptoms among the patients for the treatment of Swetha kuttam.

<b>Symptoms</b>	<b>Number of cases</b>	
	<b>Reduced</b>	<b>Not Reduced</b>
<b>Depigmentation of Skin</b>	28 80%	7 20%
<b>Depigmentation of Skin and Hair</b>	3 60%	2 40%

**Software:** spss17 version

**Number of cases:** 40

**Test:** Fisher's Exact test

**Confidence Interval:** 95%

**Result:**

**P Value (2 tailed):**  $p < 0.05$

**Inference:**

Since the p value is significant ( $< 0.05$ ), The hypothesis is not accepted. So there is significant reduced symptoms among the patients for the treatment of Swetha kuttam. Hence it is concluded that the treatment was effective and significant.

## **CONSENT FORM**

I certify that I have disclosed all the details about the study in the terms readily understood by the patients.

**DATE :**

**SIGNATURE**

**NAME**

## **CONSENT BY THE PATIENTS**

I has been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of **PUVARASAM KUDINEER CHOORNAM and NUNA THYLAM** for the treatment of Swetha Kuttam.

**DATE :**

**SIGNATURE:**

**NAME:**

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## **CASE SHEET PROFORMA FOR SWETHA KUTTAM**

GOVT. SIDDHA MEDICAL COLLEGE & HOSPITAL,  
CHENNAI – 106.

POST GRADUATE DEPARTMENT BRANCH – I MARUTHUVAM

IP/ OP NO	:	NATIONALITY	:
BED NO	:	RELIGION	:
WARD NO	:	OCCUPATION	:
NAME	:	INCOME	:
SEX	:	DATE OF ADMISSION	:
PERMANENT ADDRESS :		DATE OF DISCHARGE	:

**DIAGNOSIS :**

**TEMPORARY ADDRESS :**

**MEDICAL OFFICER:**

Govt.Siddha Medical College &  
Hospital, Chennai – 600 106.

**COMPLAINT AND DURATION :**

**HISTORY OF PRESENT ILLNESS :**

**HISTORY OF PAST ILLNESS :**

**PERSONAL HISTORY/HABIT :**



**FAMILY HISTORY:****GENERAL EXAMINATIONS**

Consciousness :  
Built :  
Anaemia :  
Cyanosis :  
Jaundice :  
JVP :  
Clubbing :  
Lymphadenopathy :  
Pedal oedema :  
Temperature :  
Pulse Rate :  
Respiratory Rate :  
Blood Pressure :

**SIDDHA ASPECT****I. YAAKKAI (udal)**

Vaatham :

Pittham :

Kabam :

Kalappu :

**II. GUNAM:**

Satthuyam :

Rajotham :

Thamasam :

### **III. NILAM:**

Kurinchi :

Mullai :

Marucham :

Neithal :

Paalai :

### **IV. PARUVAKAALAM:**

Kaar (Avani – Purattasi)

Koothir (Ippasi – Karthigai)

Munpani (Margazhi – Thai)

Pinpani (Maasi – Panguni)

Elavenil (Chittirai – Vaikasi)

Muthuvenil (Aani – Aadi)

### **V. PORI – PULANGAL (SENSORY ORGANS)**

Mei – Sensation :

Vaai – Taste :

Kan – Vision :

Mooku – Smell :

Sevi – Hearing :

## **VI. KANMENTHRIYAM / KAMMAVIDAYAM (MOTOR ORGANS)**

Kai – Dhaanam :  
Kaal – Kamanam :  
Vaai – Vasanam :  
Eruvaai – Visarkkam:  
Karuvaai – Aanantham:

## **VII. UYIR THAATHUKKAL**

### **A. VAATHAM**

Praanam :	Naagan :
Abaanan :	Koorman :
Viyaanan :	Kirugaran :
Uthaanan :	Devathatthan :
Samaanan :	Thananjeyan :

### **B. PITTHAM**

Anala pitham :  
Ranjaga pitham :  
Aalosaga pitham :  
Saathaga pitham :  
Praasaga pitham :

## **KABHAM**

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

## **VIII. UDAL THAATHUKKAL**

Saaram:

Senner :

Oon :

Kozhuppu :

Enbu :

Moolai :

Sukkilam / Suronitham :

## **IX. ENVAGAI THERVUGAL**

Naadi :

Sparisam :

Naa :

Niram :

Mozhi :

Vizhi :

**Malam:**

Niram :

Edai :

Erugal :

Elagal :

**Moothiram:**

**i. Neerkuri :**

Niram :  
Manam :  
Edai :  
Nurai :  
Enjal :

**ii. Neikuri:**

**CLINICAL PARAMETERS:**

**IST MONTH**

S. NO	CLINICAL FEATURES	DURING 1 <sup>ST</sup> DAY OF TREATMENT	PROGRESS			
			Ist Week	IInd Week	IIIrd Week	IVth Week
1	Hypopigmentation					
2	Area					
3	Shape					
4	Pruritus					
5	Erythema					
6	Depigmentation of hair					
7	Sensation					
8	Scaling					

+ = Present

- = Absent

## II<sup>nd</sup> MONTH

S. NO	CLINICAL FEATURES	PROGRESS			
		Ist Week	IIInd Week	IIIrd Week	IVth Week
1	Hypopigmentation				
2	Area				
3	Shape				
4	Pruritus				
5	Erythema				
6	Depigmentation of hair				
7	Sensation				
8	Scaling				

+ = Present

- = Absent

## III<sup>rd</sup> MONTH

S. NO	CLINICAL FEATURES	PROGRESS			
		Ist Week	IIInd Week	IIIrd Week	IVth Week
1	Hypopigmentation				
2	Area				
3	Shape				
4	Pruritus				
5	Erythema				
6	Depigmentation of hair				
7	Sensation				
8	Scaling				

+ = Present

- = Absent

### EXAMINATION OF OTHER SYSTEMS

1. Cardio vascular System :
2. Respiratory System :
3. Abdomen :
4. Central nervous system :

## LABORATORY INVESTIGATIONS

### 1. BLOOD

TC :  
DC : P : % L: % E: % M: %  
ESR :  
½ hour :  
1 hour :  
Hb (g/dl) :  
Blood sugar : F : PP:  
Blood urea :  
Serrum cholesterol :

### 2. URINE

Albumin :  
Sugar :  
Deposits :

### 3. MOTION

Ova :  
Cyst :

**FINAL DIAGNOSIS :**

**TRIAL DRUG:**

**MEDICINE:**

**I        Puvarasam Pattai Kudineer Choornam - 25 gm/60ml twice day**

**DURATION OF TREATMENT: 3 Months**

**PATHIAM AND APATHIAM**

**MEDICAL OFFICER SIGNATURE**

**H.O.D.**



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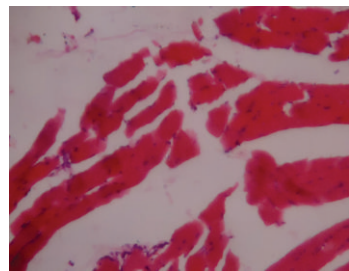
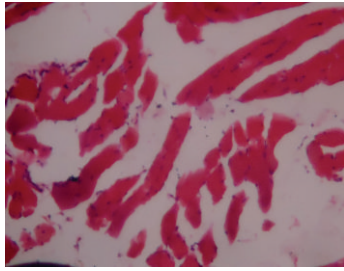
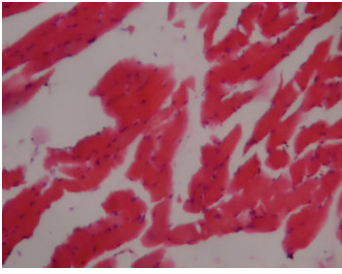
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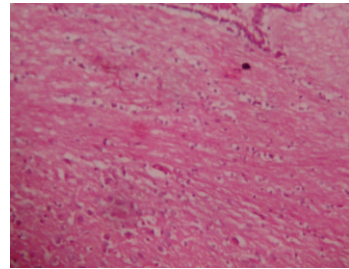
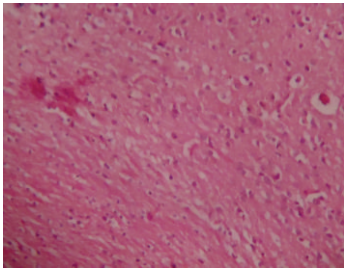
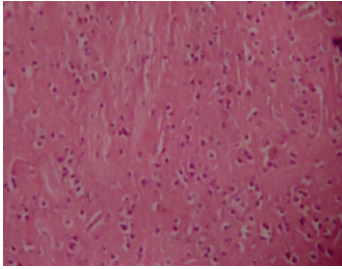
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# HISTOPATHOLOGY SLIDES

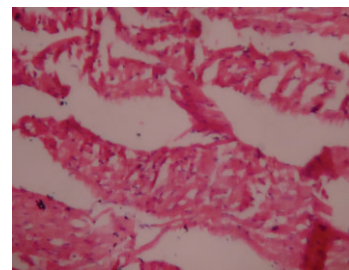
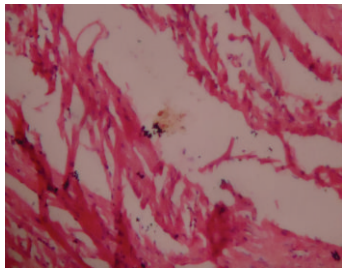
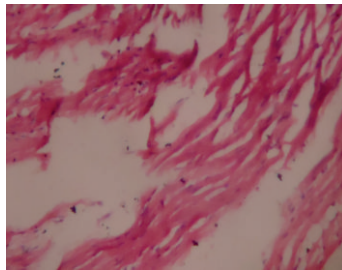
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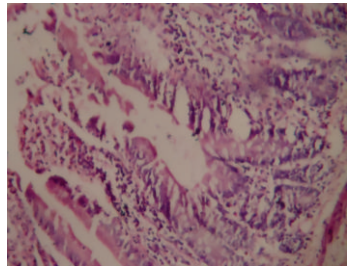
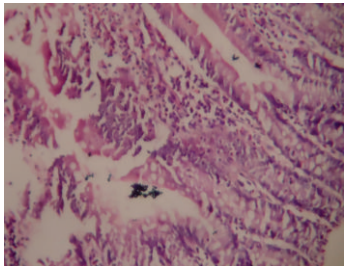
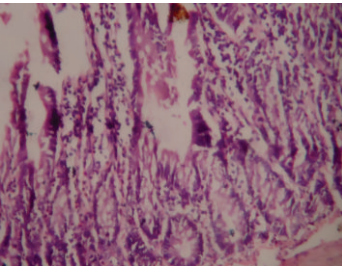
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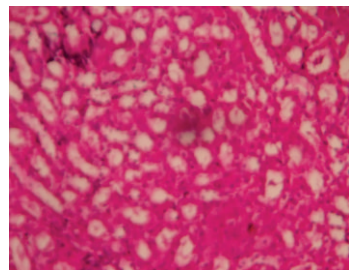
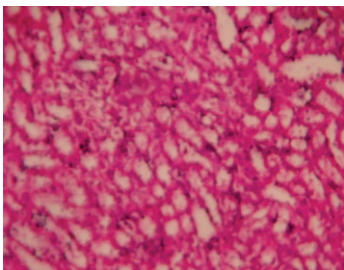
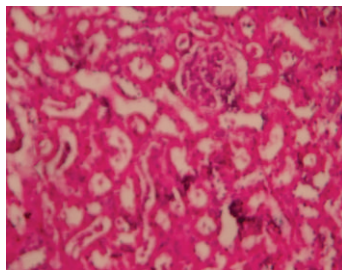
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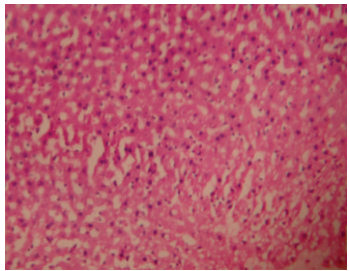
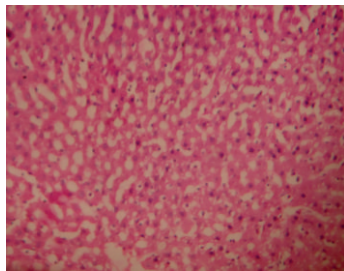
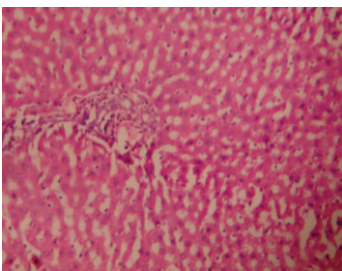
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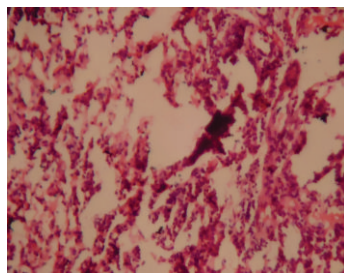
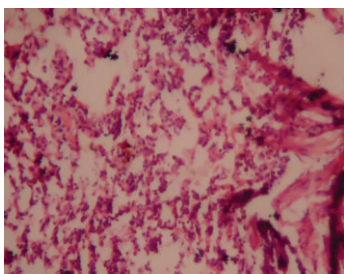
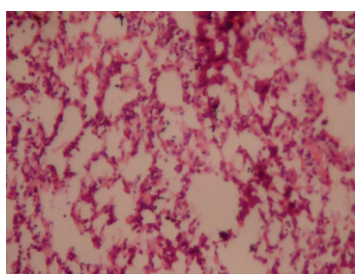


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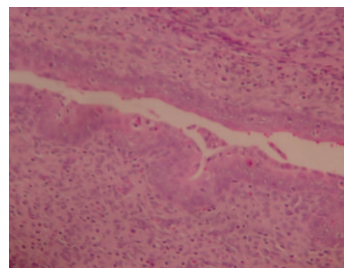
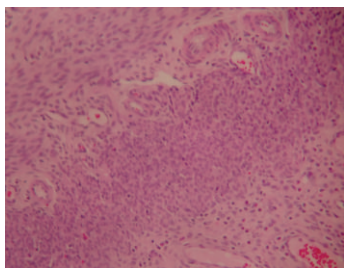
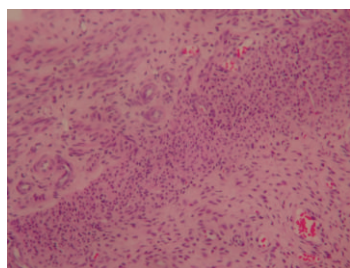




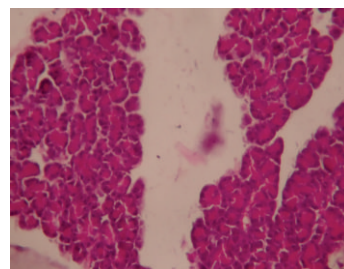
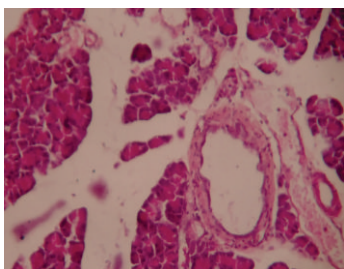
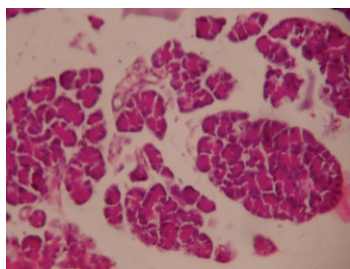
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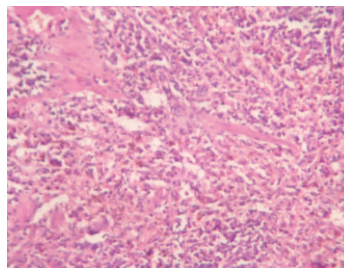
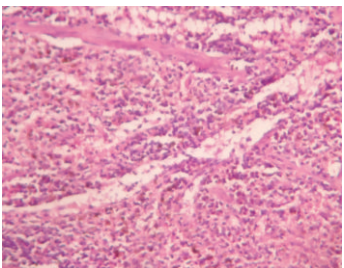
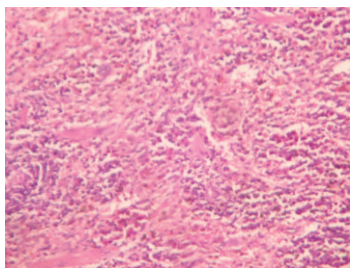
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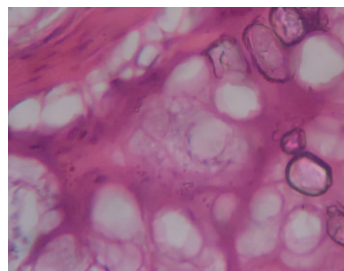
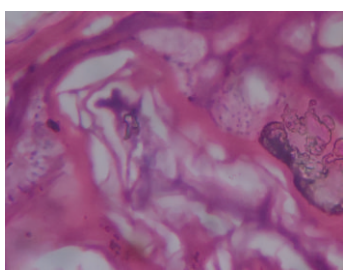
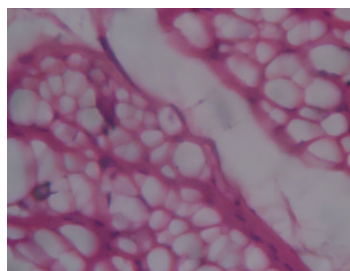
PANCREAS



SPLEEN



STOMACH



TESTIS

